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MAX A. GOLDSTEIN, M.D.

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NUTRITION AND RELATED PROBLEMS.

ISAAC H. JONES, M.D., Los Angeles, Calif.;
HAROLD S. MUCKLESTON, M.D., Pomona, Calif.;
and
JEROME A. HILGER, M.D., St. Paul, Minn.

This is our twelfth editorial concerning Nutrition. Last year we suggested that there are two distinct groups of problems which we meet in practice[1]—on the one hand the obvious presence of infection, neoplasm or trauma, and on the other hand the far less obvious effects of disturbed nutrition, endocrines, autonomic influence, stress and allergy. In studying the literature we became impressed with the close relation of nutrition with these kindred entities—the endocrines, autonomic influence, stress and allergy. It seems to us that they not only overlap but often interlock. The understanding of one is certainly not clear without the understanding of the others.

The treatment of infection, neoplasm or trauma is not difficult to determine; but we clinicians, absorbed in providing help for the patient, approach the five other problems with far less assurance. The autonomic nervous system has recently attained prominence in the minds of many, notably Hilger[2,3,4] and Williams.[5,6] It is already evident and has become recognized that its disturbed function is responsible for many lesions that we encounter. What can we do to identify and correct such lesions—and those due to disturbances of nutrition, endocrines and stress, or to allergy? In taking a history we can at least mention the five subjects to the patient, and

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see if one or more of them may lead to something suggestive. At least this gives an approach to the problem; and in the discussion of these points the patient may reveal pertinent facts. We find this helpful, not only with patients whose symptoms have no obvious cause, but particularly with those whose history and findings offer no clue whatever.

Reports in the literature on these five problems are the object of our constant search. The particular aim is to find those that suggest an approach to treatment. For the most part, these articles are from sources which our readers ordinarily would not see.

Of course Nutrition, considered and defined as the state of *health of tissue,* embraces far more than a mere study of food; however, the importance of good food itself is obvious. Through the years it has become evident that truly first-class food is simply not available, except for the very few who raise their own. The cold fact is that most people will continue to fare as well as they can on second-class supplies. Even at that, few people eat the best available food, simply because emotion and not reason is the great factor in human life, and we have become accustomed to enjoying the impoverished foods—the French pastries, the sweet soda-pop and the sugared ice-cream. We can at least suggest, or even urge, that the patient restrict the intake of everything made from white flour and refined sugar—telling him "these reduce, as it were, the high-octane of the fuel on which we travel. It is perfectly possible for our automobile to work on a gasoline of the lowest grade. Similarly, inferior food can sustain life; but for health, human fuel must be of high-octane rating."

In civilization, especially in urban life, the nutritional result is definitely not good. We are all confronted daily with problems due, wholly or in part, to lack of the right foods; for example, the "stuffy" nose, non-suppurative sinuitis, lesions of the conjunctiva, cornea, tongue and angles of the mouth, possibly nerve deafness with no apparent cause, and frequent "colds." To what extent these routine office problems may be explained by poor nutrition, with our present knowledge we can only conjecture.

ENDOCRINES.

Laboratory tests can now provide a surprising amount of new and enriching information about the status of endocrine function. Clinicians are not yet fully aware that such detailed information is available; however, the question is whether this new set of facts enables us to meet the situation and provide physiologic replacement therapy. Does the extra knowledge improve our treatment? Unfortunately, the problem is not a simple one. All would be solved if only replacement therapy could be used to make up for each deficit; but when the report comes from a modern biologic laboratory we are as yet unable to apply it adequately to the patient. With the endocrine glands and the many secretions that have been more recently investigated, we lack the feeling of assurance that we have had with the earlier triad—epinephrin, thyroxin and insulin. Epinephrin continues to be invaluable; thyroid extract, similarly, is regarded as providing good replacement therapy; and the diabetic still finds help, and at times actually his salvation, in insulin. The uses of the other endocrine secretions on which attention is now focused, seem to be fenced about by imperfect knowledge of their chemical effects. Also they are so interrelated that most clinicians feel insecure as to the end result of therapy. At best, there is an appreciable amount of uncertainty except among the very few who have the fullest knowledge.

Conscious of our limitations we sent the above paragraph to three who could, with authority, clarify the present status of hormone therapy; and in presenting their replies we feel that this has been accomplished.

Fowler[7] comments that the above paragraph simply asks for calm appraisal. Although there has been marked progress, many indications and contraindications are still to be determined.

Simkin[8] answers: *1.* In the attempt to interpret a laboratory report in terms of the clinical picture, when there are out-and-out findings there is no problem. The border-line results and the mild deviations from the norm cause perplexity. For example, he is not always able to diagnose mild hypothy-

roidism on clinical grounds, and to correlate the laboratory findings with any degree of consistency. On the other hand, myxedema is a clinical diagnosis which requires no laboratory tests at all. *2.* A blood or urine concentration of a hormone is the resultant of (a) rate of secretion of the hormone; (b) its utilization by the tissues, and (c) its excretion. We know nothing of the tissue utilization of the hormones, or what factors influence it. Thus slight abnormalities in laboratory tests may reflect changes in tissue utilization, rather than under- or over-production of the hormones by its gland. *3.* We have no clear cut criteria as to what constitutes optimal replacement therapy, quantitatively speaking. What determines adequate thyroid dosage? The B. M. R.? The blood-iodine? The clinical picture? If one demonstrates an impaired adreno-cortical response post-operatively, what constitutes optimal therapy? At any rate, not over-dosage, which may be harmful. *4.* Much is still to be learned about the tissue effects of many hormones. How do hormones affect the nasal mucosa or the auditory apparatus? *5.* Changes in one endocrine gland affect other endocrine glands. For example 50 per cent of myxedema patients exhibit laboratory evidence of adreno-cortical insufficiency, many even after adequate thyroid replacement therapy; therefore, it would appear that such patients might well receive appropriate steroid therapy, if treatment is to be complete.

Shelton[9] states that long-acting preparations are now available, which enable the experienced clinician to approximate the normal function by replacement. Naturally, the problem is not a simple one. There is a marked interrelationship among all the active principles of the endocrine glands; when coupled with heredity, environment, and the end-organ response, it makes every individual a subject for clinical research. Most people do not realize that the endocrine secretions are the fundamental factors in practically all physiologic function. Shelton further offers a helpful concept: if one will think of the hormones, when coupled with the vitamines and enzymes, as the *artisans* who put the building blocks together according to a preconceived plan (heredity), and then maintain them in equilibrium throughout life, one will

have a clear understanding of the importance of the endocrine system in the body economy. Such a concept makes one realize how important it is to consider endocrine function in every field of medicine. When one considers that the active principles of the pituitary, parathyroid, ovary, testis, adrenal, and even the pancreas and thyroid have been isolated only in the past thirty years, it is natural that the clinical evaluation of their preparations is not always clear, especially to those who do not have frequent occasion to deal with this subject.

An excess or a deficiency of cortisone or corticotropin can produce various changes, but Salassa[10] makes it clear that their precise mode of action is unknown. The limits of usefulness and the hazards are not yet defined. All that one can give is a current appraisal of a rapidly developing subject. Among the contra-indications are peptic ulcer, tuberculosis and emotional disturbances. Many massive gastric hemorrhages have occurred even when nothing suggested that the patient had an ulcer prior to the hormone treatment. Symptomatic improvement and a general feeling of well-being may occur in patients with pulmonary tuberculosis after the use of cortisone; but there is a tendency to cavitation, and a fatal outcome has occurred. Its use in most patients with tuberculosis is best avoided. In some unknown manner, cortisone appears capable of suppressing the reaction of inflammation. It seems to do this regardless of the cause. If the inflammation is detrimental, the suppressing effect of the cortisone will favorably modify the disease; but if the inflammation is of a protective nature, then the cortisone may do harm. Convincing evidence exists that the use of cortisone leads to temporary suppression of function of the adrenal cortex. Once suppression occurs, it probably is continued by even small doses and may persist for months; at such a time unusual stress as from injury, operation, burns or severe illness may precipitate acute adrenal insufficiency. Because these hormones can suppress or alter the symptoms of many diseases, it is not surprising that physicians are tempted to use them prematurely when diagnosis is difficult or delayed. Patients with undiagnosed states of fatigue, or with skeletal

or muscular pain of unknown origin, are among those who most often receive these hormones without benefit of a diagnosis. Not only is their premature use hazardous, but it may alter the clinical picture to such an extent that diagnosis is made even more difficult. It would seem unjustified except in the most unusual circumstances to use these hormones before the establishment of a diagnosis.

When corticotropin first became available in 1949 it was promptly tried in certain eye diseases. As Steffensen[11] observes, it then seemed reasonable that cortisone would give similar results; this is now well established and no essential difference has been noticed in the action of the two drugs. Cortisone was then tried by local application and many have considered it to be of great value. Some consider that hydrocortisone is physiologically more important than cortisone, and regard it as the main steroid of this type produced by the adrenal cortex. The interrelation of the endocrine glands is well illustrated in that corticotropin or cortisone inhibits thyroid function through the suppression of the thyroid-stimulating hormone from the pituitary. As this depressing effect shows up after about four weeks, it may then be necessary to give thyroid to overcome this depression of thyroid function. A most common side effect, especially undesirable in cases with cardiac and renal disease, is the retention of sodium and water, which leads to edema. If continued treatment with cortisone is necessary, diuretics may be given. Because excellent benefits in the acute ocular inflammatory diseases can frequently be obtained with a short course of these hormones, there is no absolute contraindication to their use. One should proceed cautiously in patients with hypertension, cardiac disease, diabetes, or peptic ulcer. It is recognized that cortisone tends to block the antigen-antibody reaction; this would explain its usefulness in allergic conjunctivitis, vernal conjunctivitis and possibly some cases of iritis and uveitis. It is also well known that cortisone has the ability to inhibit inflammation, allergic or otherwise. This is important even though the reasons are not yet clear.

THE AUTONOMIC.

In a study of these interrelated problems, again and again one comes upon the subject of peptic ulcer, due to autonomic disturbance, whether the origin be endocrine or stress. The subject looms large. The essential factor seems to be the autonomic nervous system; a neat picture, showing how it may play a part in peptic ulcer, is drawn by Stone.[12] Localized interference with the blood supply can occur from vasoconstrictor impulses (sympathetic) or from hypertonicity of the muscular coat (parasympathetic) which also can cause a constriction. In brief, the mucosal circulation may be reduced by direct sympathetic action, or indirectly, by the parasympathetic. Any stress or anxiety can eventuate in ulcer. The concept is that stress from any cause, may stimulate the pituitary to an increased production of corticotropin, which in turn produces an accelerated secretion by the adrenal cortex of steroid hormones that so effect the autonomic mechanism as to cause an ulcer. Some ulcers have apparently been caused by treatment with cortisone, or latent ulcers activated, or known ulcers made worse by it.

STRESS.

More and more the literature is presenting the Psyche as the center of interest—and influence. This has been particularly true during the past few years and in our ear, nose and throat literature it is emphasized to a startling extent. In referring to an article, "Memories Before Birth," it is no wonder that Merlis[13] makes an understandably sarcastic comment: "With psychiatry now entering into all phases of human life, psychologic gobbledygook has finally reached the end of the line. Et tu, Fetus?"

The emotional life surely plays a larger role than we have realized, and we recognize this, even though the treatment suggested—to "pluck from the heart a secret sorrow"—is more easily said than done. The patient has always asked, "What does it mean—mind over matter?" The popular word came to be "Psychosomatic" and when this word was first used many patients thought that it signified a new approach,

in fact was a real discovery. Of course, the only new thing about it was this new word applied to the old well-known relationship. As physicians we have always known the Captain of the ship; however, it is true that we are becoming better acquainted with the Executive Officer, the Hypothalamus.

An emotional crisis is the determining or precipitating factor in many cases of Meniere's syndrome, in the experience of Fowler and Zeckel;[14] they studied the psychologic findings in 23 patients suffering from paroxysmal attacks of vertigo, with some degree of deafness and tinnitus in one or both ears. In every case the study included a slit-lamp inspection of the bulbar conjunctiva, during the observation period; clumping of red blood cells and sluggish flow in small vessels were noted. These were assumed to be concomitant with similar changes in the blood vessels of the internal ear. Some one outstanding situation of life stress seemed to be a common factor, immediately preceding a paroxysm of vertigo. Typical stresses were: *1.* antagonism to an individual or a group of individuals; *2.* a state of tension resulting from abnormal avoidance of sex relations; *3.* conflicts centering around sexuality; *4.* the death of a near relative; *5.* emotional crises, such as fear of going insane or outbursts of anger. The authors believe that there are strong indications that certain personality types tend to develop tinnitus, and perhaps vertigo, under stress. Deafness in the early stages of Meniere's syndrome is often transient and unilateral, and may for some length of time be unnoticed by the patient.

On the long popular hypothesis that alopecia areata is a response to stress, usually emotional, it seems to Arnold[15] that the prognosis is improved by belittling the disorder, by giving a highly optimistic prognosis and by giving no local treatment. This is his impression after seeing 135 patients. The prognosis, following reassurance, is good for recovery within three months, and excellent for recovery within a year. It is interesting that the most encouraging prospect is given by those authors who assign the least value to topical medication. It is a familiar observation that medical or surgical treatment may make it more difficult to get rid of a psycho-

genic complaint; it is tempting for us to postulate that giving these complaints serious consideration lends them, in the patient's eyes, a certain element of value in that they gain for him attention and sympathy.

A good example of a mechanism over which one has no control is sweating. The average output of a typical sweat gland is 0.003 mg. per minute. Lobitz[16] mentions that sweating is largely due to parasympathetic action. Rado[17] notes that rage, frankly expressed, appears to have little if any effect upon the sweat mechanism, whereas latent rage may excite profuse emotional sweating. To correlate psychodynamics and physiology seems to Rado to be the supreme task of medicine today.

The changes in living habits, environment and stress to which an individual is exposed when called from civilian life and confronted with the emergencies and strain of war, are analyzed by von Briesen.[18] In his observations, response to the cumulative effects of fatigue varied so greatly as often to defy diagnosis. Neurologic disabilities could not be promptly typed and appraised; the organic and the functional or reversible merged one with the other. To infection, trauma, vascular disease, neoplasm and degeneration, it seems clear that stress must be added as a frequent primary etiologic agent.

ALLERGY.

Williams[19] defines allergy as a localized autonomic dysfunction of the cholinergic type. A spasm of the artery is associated with dilatation of the capillary and venule that sometimes amounts to a varicosity. Then follows anoxia of the capillary loop, with injury to the endothelium. Such anoxia results in "sludging" of the blood, local release of toxic metabolites and increased permeability of the capillary loops—with the formation of the typical allergic wheal. The organ of Corti receives all its nutrition from the endolymph, since no blood vessels supply it. For this reason it differs from organs that receive a vascular supply. This difference, however, is more apparent than real, because all cells receive their actual nutrition from lymph. The treatment which gives the most

satisfactory results is a special diet, with other measures to help in the elimination of sodium, administration of a vasodilator to decrease arteriolar spasm, and of an inhibitor to depress overactive cholinergic nerves. He has found that patients can be persuaded with relative ease to continue to use his special diet—a modification of that of Schemm.[20]

McGrath's[21] report of the occurrence at the same time of glaucoma following injury, and of Meniere's syndrome, covers the clinical course of one patient. He reviews the literature since 1934 and finds many instances of concurrent eye and ear symptoms; the presence at one and the same time of glaucoma and Meniere's syndrome is not so rare as one would suppose. Six such cases have been reported by one observer. It seems possible that these two conditions have a common angioneurotic origin.

While some children may lose their allergic reactions after puberty, Knight[22] emphasizes that they are in the minority. He views allergy as an abnormal reaction of the body cells to an ordinarily harmless substance. The tendency to allergy runs in families. The child of one allergic and one normal parent may or may not develop allergy; when both parents suffer from allergy, the probability is much greater. Allergy may be one manifestation of poor adaptation to stress. Overwhelming exposure to an allergen sometimes causes sensitization. Sensitivity to foods is likely to develop if large amounts of one food are forced, following a gastrointestinal upset. At this time the intestines may be irritated and absorb material before adequate digestion renders it harmless. The ill child should not be urged to eat. A record of the pulse rate before and after meals, as described by Coca,[23] is a new and important guide to the discovery of food allergens. A definite increase in the number of beats after contact with an allergen suggests this type of allergy.

Unfavorable reactions to penicillin occur more often than is generally recognized. Mayer, Mosko, Schutz, Osterman, Steen and Baker[24] report six instances of anaphylaxis, or shock syndrome, appearing in patients who had previously been treated with penicillin; some of these patients had re-

acted in the past, but never to an alarming degree. Reaction to the drug was immediate or appeared within a few minutes after its administration, in sweating, dyspnea, weakness, cyanosis, with pulse and blood pressure unobtainable. After the use of cardiac and respiratory stimulation and artificial respiration, all except one of the patients recovered. The patient who died had been under treatment for syphilis during eight years and had received penicillin in three courses without ill effects, but on beginning the fourth course he died a few minutes after the first injection. Intradermal tests, also, may bring on an occasional severe reaction. Every patient should be asked about previous use of penicillin; a history of a definite or questionable hypersensitivity should lead to a skin test, with high dilution of penicillin; should this prove positive, the physician is faced either with a definite contraindication to penicillin therapy or with the need of weighing the risk involved.

As a further impressive warning of the danger that sometimes comes from giving penicillin, whether by intramuscular injection, by instillation into the nasal sinuses, by aerosol stream or by oral use, Feinberg, Feinberg and Moran[25] present an illuminating review of nine instances of immediate, anaphylactic, and, at times, fatal reactions to penicillin. The symptoms may be relatively mild, such as urticaria and transient embarrassment of breathing, may be severe with evidence of shock in varying degrees, or fatal in even as short a time as two or three minutes. The use of antihistamines as a prophylactic procedure has no sound basis. The authors consider that desensitization is a very dubious means of raising a patient's tolerance; if it could be done, it would take at least several months. When more than a few days have elapsed since the last previous dose of penicillin, a skin test should be made; if this causes a positive reaction, penicillin should not be given.

Fagerberg[26] considers that to everyone working with allergic diseases it is clear that a more or less strongly pronounced psychologic element enters into them. Even the most eager allergists will not deny this. Many times this fact is observed

by the patient himself, and he may state that his troubles increase if he is nervous or feels excited. A patient who is hypersensitive to roses can get a violent attack of asthma simply by looking at a picture of roses. Before allergic phenomena were sufficiently known, the diseases to which we now give the name of allergy were thought to be nervous disorders. When Pirquet discovered allergy in the early 1900's there was a turn in the opposite direction. It was found, or at least believed, that one disease after another for which no genesis had been known before, could be classified as allergy; but it soon became evident that the problem was not a simple one. The author selected thirteen asthmatics, none of whom presented an obvious explanation. He found that psychogenic factors played a certain and often very manifest part.

* * * * *

A talk on what he terms "The Common Ground of Allergy, Autonomic Dysfunction and Endocrine Imbalance,"[27] was made by one of us (J.A.H.), and is presented here in full:

This presentation offers nothing new or strikingly original. Rather it is intended to offer a perspective—a viewpoint which can make the approach to some of our common clinical problems simpler; a viewpoint correlated with the ultimate goal of intelligent therapy; a viewpoint which has at least the merit of integrating current attitudes stoutly defended each by its individual proponent. There is danger in over-simplification; but this danger should not discourage one from efforts to reduce problems to basic terms.

Medical specialism can be viewed from two standpoints: the one embraces familiarity with a restricted segment of the body and the influence of the functions and disorders of that segment upon the organism as a whole; the other perspective views the whole organism and the influence of its functions and disorders upon the restricted segment. The future and usefulness of medical specialism depends upon the willingness of the specialist to accept the latter viewpoint. Medical methods which dismember the patient can only establish a bizarre

hodge-podge of medical knowledge which, from divergent angles, resembles a cubist's nightmare rather than an orderly system of facts and theories related to the whole human being. Witness, for example:

If a young woman in the first trimester of pregnancy describes a recently acquired and persistent nasal obstruction and mucoid nuisance to her obstetrician, he recognizes it as a not uncommon endocrine imbalance which, if untreated, may improve as the pregnancy proceeds, or may persist until the early post-partum period. A psychiatrist, on the other hand, might be pardoned for suspecting an emotional conflict, perhaps allied to her pregnant state, as the source of her nasal problem. The rhinologist versed in nasal allergic problems and their management might well indict the dust inhalant features of the rug and pad in the new living location. It is obvious that this young woman has a definite etiology for her individual problem which is not going to vary with the clinical threshold she crosses. Her differential requires a broad view of her as a total being and not a package of parts.

Allergic reactions, states of autonomic dysfunction and of *endocrine imbalance* can produce identical clinical symptoms. In the field of otolaryngology they are commonly expressed as *rhinitis, vascular pain, myalgia (cervical or pharyngeal)* or *inner ear disturbance* with variable degrees of *tinnitus, hearing impairment* and *vertigo.* The common denominator in this wide range of expression is *abnormal extra-vascular fluid.* It creates the immediate problem in the end organ. The *method of its formation* in the precipitor states of allergy, autonomic dysfunction and endocrine imbalance *may vary* but the end result in local symptoms is the same when excessive extravascular fluid is established in the end organ tissue.

Of all the peripheral tissues involved in extra-vascular fluid imbalances, these of otolaryngology, as noted above, are amongst the most frequent; therefore, the otolaryngologist must equip himself with the differential knowledge to seek the specific cause in the individual case or he must equip himself with the knowledge that a differential exists and someone

qualified must seek it out. Results are not obtained by a rigid approach to the problems of transudate through the narrow and limited view of allergy alone, or of endocrinology alone, or of psychosomatic concepts alone.

It is the purpose here consciously to avoid exhaustive pro and con bibliographic references to the mechanisms of transudate formation in these three states—allergy, autonomic dysfunction and endocrine imbalance. There is much that is known of these mechanisms and much more that is not. The purpose of this discussion is better served by concise references to accepted fact and theory. In addition one must maintain an open and receptive mind because the medical advances of tomorrow will inevitably modify the facts and theories of today. Additional etiologic factors in transudate formation will be brought to light to further clarify today's incomplete differential.

The state of altered reactivity or allergy in a confined sense implies the presence in the shock organ of the allergic individual of anti-bodies in intimate association with smooth muscle cells—principally of the arteriolar bed. The anti-bodies are an immunologic response to antigenic stimulation. There is evidence that this immune reactivity is mediated through the basal nuclei of the brain—principally aggregated in the hypothalamic region. The anti-body, once established in association with the smooth muscle, stimulates the latter to spasm when the organism is re-exposed to the originating antigen. Possibly from the resultant ischemic hypoxia and starvation of its endothelium, the capillary tube becomes abnormally permeable and fluid exudes into the extravascular spaces. This sequence is widely accepted as the method of transudate formation in the allergic reaction.

Autonomic balance is mediated through the basal nuclear aggregates of the brain stem. In this central switchboard the neurovascular, neuromuscular and the neurosecretory activities of the vegetative nervous system are integrated. Reflexes from body surface receptors stimulated by heat, cold, light, barometric variations, etc., and impulses from higher psychomotor areas play through these centers upon the autonomic

activity of peripheral organs. Unbalanced expression in peripheral organs results when integration is faulty. Faulty neurovascular impulsion has its principal influence on the arteriolar bed. Here sphincteric spasm indistinguishable from the antigen-antibody conflict of the narrow allergy can result in similar damage to the capillary endothelium and capillary leak. The resulting transudate problem is indistinguishable from that of the narrow allergy.

Endocrine imbalance has a less well defined mechanism of transudate. The thyroid, the gonadal and the adrenal cortical hormones all are important to the proper position of body fluids. In endocrine variations from the normal the three possible levels of endocrine fault must be considered. Of the first or primary mediator of the pituitary little is known. It is considered to be a direct or indirect stimulus pathway from the basal nuclei to the anterior lobe of the pituitary. The second level is the trophic hormone level of the pituitary itself. And the third level is the level of the target gland—principally, in fluid balance considerations, the thyroid, the gonads and the adrenal cortex. Outspoken, gross signs of endocrine imbalance are the exception, not the rule. Evidence of endocrine imbalance must be sought for. Less-than-myxedema grades of hypothyroidism present no standard complex of clinical signs. A transudate problem warrants thought of thyroid deficiency as a possible cause. The concurrence of transudate problems and the menopause or the male climacteric is suggestive evidence of deficient estrogen or testosterone source. The cyclic premenstrual accumulation of extravascular fluid with the premenstrual symptoms of headache, bloating, endolymphatic hypertension or pharyngeal myalgia is the best evidence of the relation of high estrogen levels to the phenomena. These many abnormal endocrine-caused shifts in fluid balance may result through one or several mechanisms. Little is known of them. They may include changes in the capillary endothelium itself, shifts in electrolyte balance between vascular and extravascular spaces, and tissue change such as the increase in the subcutaneous mucopolysaccharides of myxedema. Whatever the mechanism, transudate is the end product.

The patient with an abnormal extravascular fluid problem above the clavicle, and excluding the eye and the brain, is prone to gravitate to the otolaryngologist. The clinical syndromes of rhinitis, myalgia, vascular pain and labyrinthine ischemia and transudate are not difficult to recognize if one is willing to honor suppuration only when it truly exists and to cast out vague references to meaningless atypical neuralgias and pigeon-hole headaches. Once the presence of a transudate syndrome is established, it is up to the otolaryngologist or his reference physician to determine the relative responsibility of allergy, autonomic dysfunction or endocrine imbalance. The problem is not made easier by the possibility of an interplay of all three causes. This is particularly apparent when it is noted that all three are related directly or indirectly to the mediating influences of the basal nuclear aggregates. Faulty function in this central area may be due to the inherent inadequacies of a poor family brainstem or the acquired inadequacies of a post-poliomyelitic, post-encephalitic, or post-arteriosclerotic basal nuclear change. It is conceivable that this faulty function can be evidenced through all three transudate sources . . . the immunologic mediation of allergy, autonomic impulsion directly, or the primary mediation directed from the brainstem to the trophic producing pituitary —all in the same individual. Thus, a youthful hypothyroid can also manifest specific inhalant or food sensitivity. Desensitization or elimination is only partially corrective. Addition of a proper amount of thyroid substance can make him symptom free. Thus, a woman at the menopause, and in the absence of extra-ovarian estrogen sources to tide her through the critical period, may the more readily develop a transudate problem in her inner ear under the stress of physical exposure or severe emotional strain. Thus, a recurrent vascular head pain occurring cyclically in the immediate premenstrual period and additionally, on occasion, after ingestion of a particular food, or exposure to an annoying draft, or an emotional upset is a clinical commonplace. Thus, the sudden onset of acoustic and vestibular symptoms usual with an abrupt increase in endolymphatic fluid pressure may be attributed by the victim to a stressing emotional upset, severe chilling of the body through physical exposure, or the gluttonous ingestion of a

dozen hard-boiled eggs of which he is fond in spite of previous unfortunate experiences with them. Any or all of these factors might well be only the final transudate straw in the victim who was unknowingly significantly hypothyroid. It would be an interesting study to call back a large labyrinthotomy series to check the basal metabolic rates and blood cholesterols. Hypothyroidism is only a single unbalanced endocrine substrate possibility.

Is it enough then to treat the results of transudation in terms of vascular pain, rhinitis, myalgia or inner ear syndromes in a standard, predetermined manner with hormones or dehydration therapy, with elimination diet or histamine, with pharmaceuticals or psychotherapy? One suspects not. Better results can more reasonably be expected by a sensible correction of all causative factors in the individual case than by the stereotyped approach indicated by the sign on one's door.

One of the universal truths about transudate problems is this. The peripheral expression, whether nasal, acoustic, vestibular or painful, tends to recur and produces sufficient distress to add fatigue and emotional stress to the problem as a result instead of a cause. An understanding doctor with human insight is an excellent psychotherapist. If he can bring a knowledge of specific reactivity, endocrine inter-relationship and the pharmacy of stress to the problem additionally, he is ideally suited for the work. Above the clavicle the man with whom the problem is first placed is commonly the otolaryngologist. He is equipped to examine the respiratory tract, to evaluate the ear, to rule out suppurative sources and, in all ways, to complete the examination of the transudate problem. He may content himself with identification. If not, he must then equip himself to deal with the differential of transudate —to correctly balance the etiologic possibilities in every case between specific allergy, autonomic dysfunction, or endocrine imbalance. The aim of medical progress is, after all, more satisfactory result for the patient even though it batter down the protective ramparts of narrow specialism.

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ITEMS ON NUTRITION.

The science of nutrition may be said to have developed during this century. Cowgill[28] outlines its growth from the conjectural, groping stage to the present systematized study, as illustrated by the discovery that the value of liver in treating pernicious anemia lies in the presence of folic acid and "B_{12}." It seems reasonable to expect future discoveries to compare favorably with those of the past fifty years.

Because proteins are the chief constituents of all body tissues, play leading roles in their metabolism, protect against antigen reactivities, provide for wear and tear replacements and for reproduction of the species, Albrecht[29] sees the increasing importance of proteins to our thinking. Protein alone is the substance through which life flows. The lowly microbe, likewise, is assuming increasing importance as a factor in the cycle of protein metabolism. It is gratifying to learn not only of the use of antibiotics for man's protection against microbes, even to the dangerous extent of sterilizing the entire intestinal tract, but also the surprising turn from the fighting to the courting of microbes. For example it is these "good" microbes that produce better nutritional values by increasing soil fertility.

For the past twenty years emphasis has been placed upon salt and water metabolism in the search for etiology and therapy in Meniere's syndrome. Under hospital conditions Perlman, Goldinger and Cales[30] observed fifteen patients, whom they divided into two groups. In the first group they lowered the blood levels of sodium by restricting its intake and by giving ammonium chloride and a mercurial (thiomerin). In the second group extra dietary salt was given and its retention brought about by the use of desoxycorticosterone. Despite the depletion of serum sodium in the first group, several of the patients suffered severe attacks of vertigo; moreover, when their electrolytes were allowed to return to normal, their auditory and vestibular symptoms improved. In the group with elevated serum sodium, no acute attacks of vertigo occurred, nor was there a consistent deterioration of cochlear function. The authors infer that present therapy aimed at electrolyte alterations is probably ineffective.

Hypervitaminosis-A, though rare, is not a new or modern disease. The livers of polar bears contain "A" in large amounts, and Knudson and Rothman[31] report the poisonous effects that have long been noted by Eskimos and travellers. The effect of prolonged overuse of "A" on bone growth in childhood is well shown by Reyersbach, Hanelin and Joplin[32] in their report of the case of a three-year-old girl. Owing to suspicion of rickets the child received one teaspoonful of oleum percomorphum (240,000 units of "A" daily) for nine months. She became irritable, her skin was thickened, with widespread erythema, and her lips became dry and cracked. The liver was not enlarged. The vitamin-A blood level (normal 0.3 to 1.0 unit per cc.) was 26.7 units per cc. Her long bones showed periosteal overgrowths and hyperostoses.

The question arises whether penicillin has the same effect on the intestinal bacteria that create vitamins as have aureomycin and chloramphenicol. An answer is given in an editorial in the Journal of the American Medical Association.[33] Although penicillin administered by mouth produces profound changes in the intestinal flora, this effect is short-lived. Within a few days the members of the intestinal flora that have been suppressed usually begin to grow again, so that, even though the penicillin may be continued, the organisms in the bowel are much the same as before. For this reason, penicillin would not be suspected of having much effect on the bacteria that elaborate vitamins. Furthermore, the only vitamin of importance that is produced by the intestinal flora is "K." This is very different from the situation in herbivorous animals in which various bacteria elaborate various components of the B-Complex. Unlike aureomycin, which may produce increased urinary excretion of riboflavin, penicillin does not increase the loss of any vitamins in the urine. When penicillin is given in the form of lozenges or troches, acute glossitis may appear, but not when the penicillin is swallowed. It would not appear necessary, therefore, to give vitamins to patients who are receiving penicillin orally, even for long periods.

Hall[34] echoes the experience of every physician in that too often the patient's first question is not "what is the matter with me?" but rather "Should I take sulfa or penicillin?" Aside from the inconvenience, loss of time and unnecessary expenditure of money, the use of antibiotics without strict indications is objectionable from several points of view. *1. False sense of security.* The use of antibiotics may give the false impression that everything is being done that can be done for an obscure illness. *2. Development of bacterial resistance to antibiotics.* Coliform bacteria have been observed to undergo a 50,000-fold increase in resistance after only 48 hours of exposure to streptomycin. Many of the patients in Sanatoria today harbor streptomycin-resistant tubercle bacilli. Antibiotic resistance has been a frequent problem in staphylococcic infections. Staphylococci seem peculiarly prone to develop resistant variants. There is evidence that staphylococci are becoming more frequently and more markedly resistant to penicillin year by year. *3. Side effects and hypersensitivity.* A laparotomy in one patient revealed over 1000 cc. of clear yellow peritoneal fluid with marked dilatation of the serosal blood vessels and severe ecchymoses. This was interpreted as an allergic enteritis. The astounding increase in production of antibiotics has led to the very desirable reduction in cost, but has also led to indiscriminate use by some people. An important result has been the not infrequent conversion of a minor illness into a major disaster.

The tongue may well serve as an index of what is going on in the stomach and bowel, but not yet are all signs completely understood. Certain abnormal states of the oral mucous membrane, which in the past have been described as the companion of pernicious anemia and various vitamin B-Complex deficiency states, have not always been completely relieved by the therapy suggested by these states. More specifically, a parallel relationship between atrophic or inflamed oral mucous membrane and achlorhydria has been observed. Sharp[35] considers that certain of these states in the oral mucous membrane are indicators of abnormal gastric function. The determination of achlorhydria has been much impeded in the past by the great discomfort to the patient of passing the stomach

tube. Sharp has simplified a tubeless gastric analysis, first described by Segal and his associates and called the "cation-exchange-indicator-quinine" (C.E.I.Q.) urine test. This does not require the use of intubation for withdrawal of stomach contents, but by urine examination it provides a means of measuring its hydrochloric acid production. After simultaneously performing the traditional gastric intubation and the C.E.I.Q. test on one hundred patients, he concludes that the latter test is the more accurate. In this series, thirty patients were found to be achlorhydric. Their symptoms were burning or rawness of the tongue, with or without dryness of the mouth and throat; and their signs were frequently an erythematous appearance of the tongue, atrophy of the filiform papillae, and not infrequently a geographic type of tongue. Hydrochloric acid is given by means of a specially prepared tablet, containing betaine hydrochloride, methyl cellulose and pepsin, which delivers hydrochloric acid over a physiologic time-interval. Improvement in symptoms, as well as a return to a more normal mucous membrane, was frequently attained. Ivy[36] states that gastric acidity slows up after age 20; achlorhydria increases considerably after 40; and by 65, 35 per cent of people do not secrete acid at all.

As compared with the beginning of this century there are now twice as many people in this country, and four times as many old people. "Old" means age 65 or older. Of these 13 per cent are 80 or older with a life expectancy of over 5 years. Ziffren[37] feels that necessary surgery should not be withheld from these old people. The results are so much better now, largely because of the nutritional care before and after operation and the availability of antibiotics. If there be no emergency, time must be taken to be sure that the blood volume and the protein reserve of the patient are adequate and that he is properly hydrated. It is easy to be misled by a presumably normal hemoglobin and blood count in the elderly, for it has been demonstrated that practically all elderly surgical patients have a deficiency in blood volume. The possibility of shock is very much greater in the aged person with a low blood volume. The elderly person is also very likely to have nutritional deficiency, especially if he has a

condition requiring surgery. Consequently, a nutritious diet must be provided, rich in protein and supplemented with vitamins. A supplementary formula of high protein and caloric value is given—three tablespoons of skim milk powder in a glass of skim milk. This provides 18 gm. of protein and 26 gm. of carbohydrate. This may be flavored with chocolate or vanilla and well mixed in a food blender. If needed, intravenous food can be given, but the fluid must be administered slowly to avoid overloading the circulation. The water requirement can be easily met and a major portion of the caloric needs supplied by use of the protein hydrolysates. Alcohol is usually added to these solutions to provide additional calories. The loss of the patient's protein reserve can be reduced to a minimum in this fashion. The one factor that has reduced the mortality in these elderly patients has been the marked restriction of salt and fluids administered parenterally. The elderly person has a lower metabolism and eliminates much less waste in his urine than does a younger person, and consequently requires much less fluid daily; in fact the giving of a large amount of fluid may prove actually hazardous, unless the patient is definitely dehydrated. The patient is not permitted to remain recumbent constantly; is urged to go to the bathroom and to walk about freely. Every effort must be made to have the patient out of bed and in a chair on the day of operation. If the patient can take fluids by mouth, potassium deficit is no problem, as it is easily corrected by an ordinary diet rich in bouillon broth and fresh fruit juice. Penicillin is given routinely to prevent pulmonary complications from infection. It is well to remember, however, that pulmonary complications ordinarily are preceded by edema, which can usually be prevented by restricting the salt intake and by not overloading the circulation with large quantities of intravenous fluids.

MINERALS.

So essential to the nutrition of the cell, minerals can also serve as isotopes. The use of radioactive phosphorus as a means of diagnosis of tumors of the eye has proved of value in the experience of Town.[38] He administers 500 millicuries of P^{32} in an isotonic solution of sodium chloride intravenously,

and one-half hour later he measures the uptake count, with a Geiger counter in contact with the sclera for one minute; the counter is held approximately 10 mm. from the limbus at four sites in each eye. A gross disparity in the counts, the higher count being obtained in the affected eye, he considers to be indicative of tumor tissue. In cases of retinal detachment, not of neoplastic origin, an inequality of low degree is reassuring as evidence of the absence of tumor tissue.

The retrosternal toxic thyroid gland is not definable by X-Ray, because its density and that of adjacent normal mediastinal contents are so closely similar as to cause the shadow of one to merge into that of the other. Reynolds, Corrigan and Hayden[39] report on the application of radioisotopes and the Geiger counter to this and allied diagnostic problems. During the past six years, 2046 cases have been studied. Astonishingly small adenomas and malignant nodules of the thyroid can be identified and mapped, and invaluable help given for the surgical approach. In the hands of thoroughly trained examiners a high percentage of successful tracer studies has been attained; in fact the net amount of error was only seven-tenths of one per cent.

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FUNCTIONAL AND HISTOLOGICAL FINDINGS AFTER OBLITERATION OF THE PERIOTIC DUCT AND ENDOLYMPHATIC SAC IN SOUND CONDITIONED CATS.*†

HAROLD F. SCHUKNECHT, M.D.,‡
Detroit;
and
ROBERT S. KIMURA, B.A.,
Chicago.

This experiment was designed to study the effect on hearing and on inner ear morphology, of obliterating the periotic duct§ and endolymphatic sac. Operations to obliterate these structures were performed on a series of seven sound conditioned cats.

A preliminary report of some of the early findings in five of these sound conditioned animals appeared in a previous publication.[1] Also presented in that paper were the histological findings in four cats in which Lindsay and Neff had performed operations to obliterate the endolymphatic sacs. In that paper it was stated that "the evidence indicates that the animal ears suffered no histological or functional change by losing the services of the periotic duct or endolymphatic sac." The studies have now been completed on the sound conditioned cats. It is the purpose of this paper to present the findings of the experiment in full.

PROCEDURE.

For each of seven healthy adult cats the function of one ear was destroyed by introducing a probe into the labyrinth through the round window membrane. The animals were

*This research was done in the Division of Otolaryngology of The University of Chicago.

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‡Now at Henry Ford Hospital, Detroit, Michigan.

§The term "periotic duct" refers to the channel of loose connective tissue and contained fluid which lies within the cochlear aqueduct.

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then trained to respond to pure tone auditory stimuli by moving forward in a rotating cage to avoid shock. Audiograms were determined using this conditioned avoidance response as an indicator of hearing. The method has been described in detail in another publication.[2]

Cats 1 and 2 were subjected to the operation for obliteration of the periotic duct and Cats 3, 4 and 5 to destruction of the endolymphatic sac. Both operations were performed on Cats 6 and 7, with intervening intervals of one and one-half and two months respectively.

The periotic duct was approached through the auditory bulla. A cap of bone was carefully removed from the wall of the bulla so that it could be wired back into its original position at the completion of the operation. Mucosa was elevated so as to expose bone in the area immediately pos-

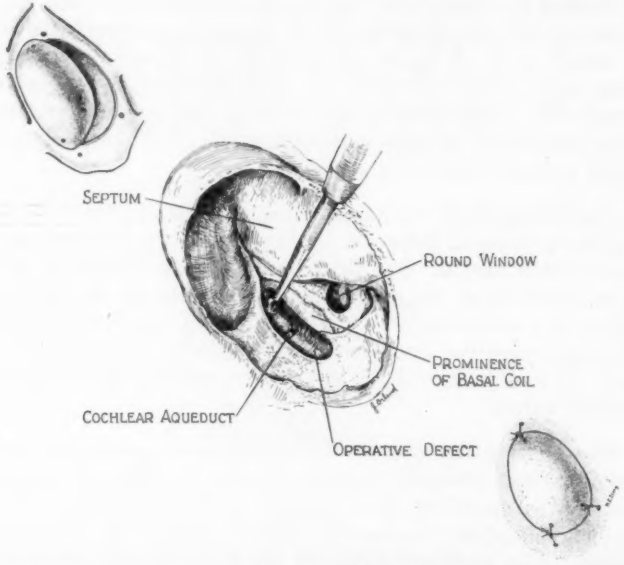

Fig. 1. Operative approach to the cochlear aqueduct of the cat. Upper left: Removal of cap of bone from the auditory bulla. Center: Opening into the cochlear aqueduct is made with a small burr. Lower right: Bony cap of auditory bulla is wired back into its original position.

teromedial to the round window. By using a small burr to remove bone, a trough was made and deepened until the periotic duct was opened, as shown in Fig. 1. The duct was opened and bone dust packed into it. The cochlear vein was torn in several ears. In three ears a viable mucosal flap was placed into the bony defect. The bony bulla cap was wired into place and the soft tissues of the neck were closed in two layers.

The endolymphatic sac was exposed and destroyed by an approach through the posterior cranial fossa (see Fig. 2). A paramedian scalp incision was made and the muscles reflected away from both sides of the lamboidal ridge. Parts of the parietal, occipital and temporal bones overlying the posterior fossa were removed. That part of the cerebellum overlying the posterior part of the medial surface of the petrous bone was removed. The small niche on this surface indicated the location of the endolymphatic sac. Both the intradural and subosteal parts of the sac were destroyed with a small burr. A short segment of the endolymphatic duct was also destroyed; however, it was necessary to leave a small part of the duct in situ to avoid entering the vestibule. The temporalis muscle was sutured over the defect in the skull and the scalp was closed.

Beginning three weeks after each operation, tests of hearing were made at intervals of three to four weeks during the observation period. The final postoperative threshold values were established by averaging the results of several tests taken on consecutive days at the end of an observation period. The postoperative audiograms were plotted in terms of the gain or loss in threshold sensitivity relevant to the preoperative values.

The observation periods following each operation varied from one and one-half to seven months for different animals. Cat 1 was sacrificed four months after obliteration of the cochlear aqueduct because of urethral obstruction by urinary calculi. The auditory threshold for five cats were essentially normal following these operations for periods of time varying from one and one-half to seven months. Because it seemed

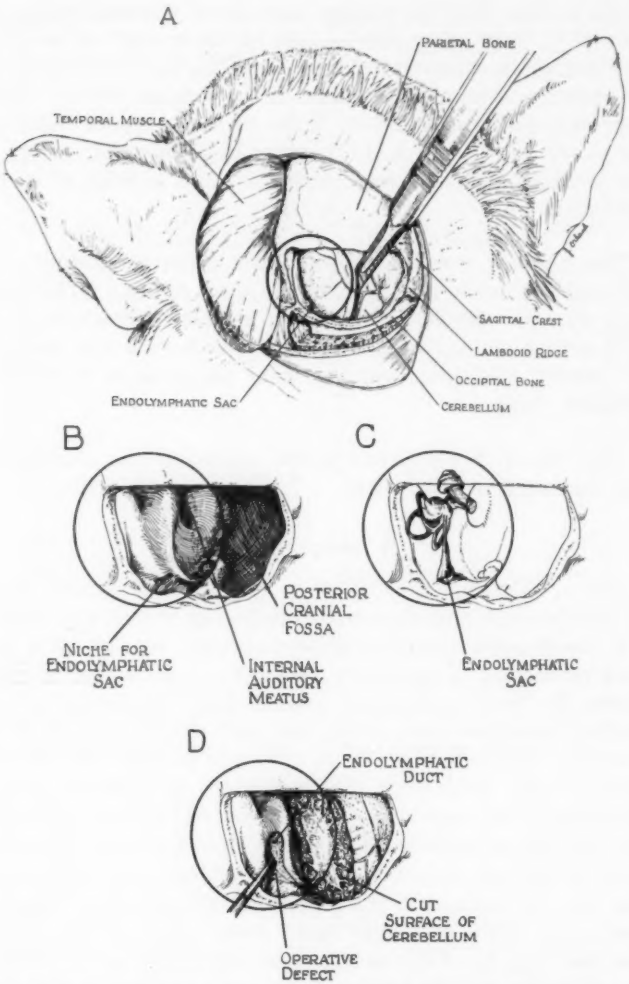

Fig. 2. Operative approach to the endolymphatic sac of the cat. A. Exposure of posterior cranial fossa and cerebellum. Part of the cerebellum is removed. The circle indicates the areas shown in the drawings below. B. Bony anatomical detail showing niche for endolymphatic sac. C. Schematic drawing showing approximate location of membranous labyrinth including endolymphatic sac. D. Small burr is used to destroy the endolymphatic sac and part of its duct.

fairly certain that the sensory and neural elements were still normal in these ears, and because of the investment of time and energy in conditioning these animals, they were used for an experiment on partial section of the cochlear nerve.[3] These five animals were sacrificed three to seven weeks following nerve section. Cat 7 had a hearing loss after obliteration of the periotic duct and was sacrificed three months after the operation.

The animals were sacrificed under general anesthesia by arterial perfusion with Heidenhain-Susa solution. The inner ears were serially sectioned at a thickness of 20 μ and every fifth section stained with hematoxylin and eosin and mounted for study. Additional sections were prepared as needed for detailed study.

The organ of Corti and spiral ganglion was graphically reconstructed for each ear.

RESULTS.

Cat 1. The function of the right ear was destroyed by labyrinthotomy and during the following month the animal was sound conditioned to auditory stimuli. An operation was then performed to obliterate the periotic duct of the left ear. Using the bone engine and a small burr the channel of the cochlear aqueduct was widely exposed. There was bleeding from the cochlear vein. Bone dust was packed into the surgical defect. Following this procedure the animal had a spontaneous nystagmus to the right and ataxia with falling to the left, which subsided completely after six days. The final hearing test taken four months later showed a 15 db hearing loss for 250 c.p.s., but the thresholds for all other frequencies tested (62.5 to 16,000 c.p.s.) were within 10 db of normal (see Fig. 3). Four months after operation on the cochlear aqueduct the animal developed an acute urethral obstruction from multiple urinary calculi. Urethral catheterization was unsuccessful. The animal was sacrificed by arterial perfusion with Heidenhain-Susa solution.

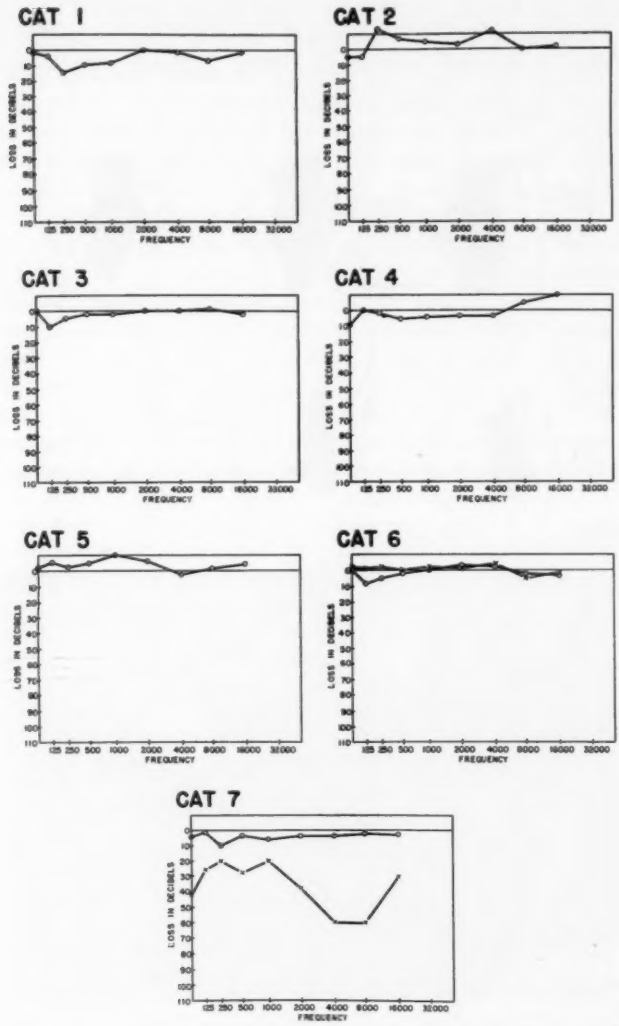

Fig. 3. Behavioral audiograms of the seven sound conditioned animals. In each case the graphs show the gain or loss in threshold sensitivity between the preoperative and postoperative tests.

Histopathology. Histological examination revealed the cochlear aqueduct and its contained periotic duct to be blocked by new bone and connective tissue. Camera lucida draw-

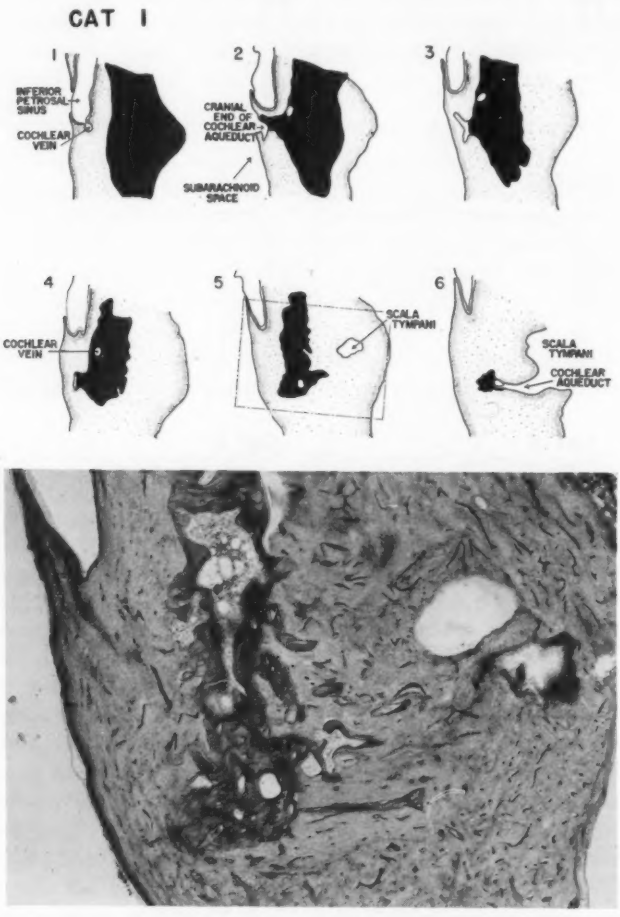

Fig. 4. Top: Camera lucida drawings from six microscopic sections in the region of the cochlear aqueduct of Cat 1. Black represents bone and connective tissue filling the surgical defect and blocking the periotic duct. The cochlear vein passed through the surgical scar to empty into the inferior petrosal sinus. Bottom: Photomicrograph of drawing "5" above.

ings and a photomicrograph in Fig. 4 demonstrate these changes. The cochlear vein had reformed as a channel of normal size passing through the bone and connective tissue of the surgical defect to empty again into the inferior petrosal sinus. No connection could be found between the cochlear vein and the veins of the middle ear mucosa. Throughout the cochlea Reissner's membrane was depressed (toward the basilar membrane) (see Fig. 5). In a large region of the basal coil Reissner's membrane was lying directly on the tectorial membrane. The tectorial membrane in turn appeared to have pressed upon the organ of Corti so as to flatten it slightly. Hair cells were present throughout. In both the lower basal and lower middle turns there were regions in which the outer hair cells were flattened and had a pale staining granular cytoplasm. Hensen's cells were markedly flattened throughout the cochlea, and in a large part of the basal coil the cytoplasm of these cells stained poorly even in areas where the outer hair cells appeared normal. Through-

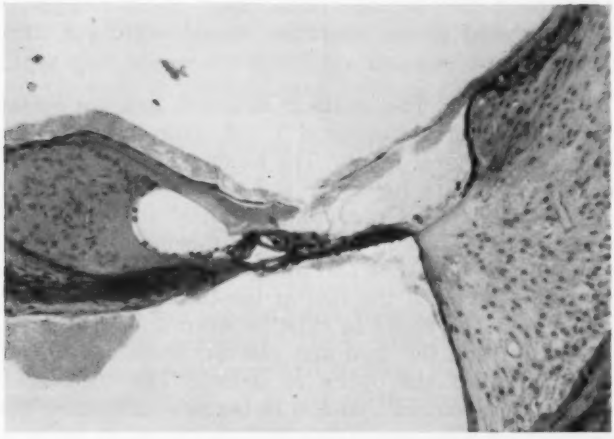

Fig. 5. Photomicrograph of organ of Corti in basal coil of Cat 1. Reissner's membrane is collapsed onto the tectorial membrane: Hensen's cells and the inner and outer sulcus cells show a marked degenerative change. There is a granular precipitate in all three scalae. The hair cells and nerve fibers are normal. This animal had nystagmus and ataxia for six days following surgery and the pathological changes are thought to represent an arrested serous labyrinthitis.

out the basal coil a layer of fine granular precipitates was seen in both the scala tympani and scala vestibuli. Smaller amounts were present in the cochlear duct. These findings are consistent with an arrested serous labyrinthitis and explains the vestibular upset which this animal experienced following surgery.

The walls of the saccule, utricle, ampullae, and membranous canals were partially collapsed inward. The maculae and cristae appeared normal. The cochlear and vestibular nerves and their ganglia appeared normal. Rasmussen's olivo-cochlear bundle was intact.

Cat 2. A left destructive labyrinthotomy was done and during the following month the animal was sound conditioned. A surgical procedure was then performed to block the lumen of the right periotic duct. The auditory thresholds were normal seven months later (see Fig. 3). Two operations for partial section of the cochlear nerve were then performed three weeks apart, following which the animal was profoundly deaf. The animal was sacrificed by arterial perfusion nine months after the operation to obliterate the cochlear aqueduct.

Histopathology: The cochlear aqueduct and its contained periotic duct were intercepted 1 mm. from their vestibular ends by the surgical lesion. The lesion consisted of vascular connective tissue in its center and new bone at its margins. Every section in this region was stained and mounted and camera lucida drawings were made to define better the relationship of the new bone and connective tissue to the periotic duct and to establish the fate of the cochlear vein. No connections could be found to exist between fluid spaces of the periotic duct and the fluid and vascular spaces of the tissue which had filled the operative defect. The cochlear vein connected with venous channels in the new connective tissue, which in turn were continuous with the veins in the mucosa of the auditory bulla. The cochlear changes were typical of those resulting from injury to the cochlear artery, which presumably had occurred during the operative procedure to partial section the cochlear nerve. The vessels within the

modiolus were severely degenerated and obliterated. The spiral ganglion appeared normal in the basal 4 mm. of the cochlea, after which there was progressively severe degeneration to complete loss of ganglion cells at 6 mm. A few inner hair cells remained in the basal 4 mm. of the cochlea, but in the remainder of the cochlea the organ of Corti was missing. In the apical 10 mm. of the cochlea the fluid spaces of the scalae were almost completely replaced by connective tissue and in several areas by a small amount of new bone. The vestibular labyrinth showed similar severe degenerative changes. The fibers of Rasmussen's (olivo-cochlear) bundle were degenerated throughout.

Cat 3. A left destructive labyrinthotomy was performed following which the animal was sound conditioned for auditory testing. The endolymphatic sac and part of the endolymphatic duct of the right ear were then surgically removed. The auditory thresholds remained normal during the subsequent six weeks (see Fig. 3). Two operations for partial section of the cochlear nerve were then performed three weeks apart. After the second nerve section the animal was profoundly deaf and one month later was sacrificed by arterial perfusion with Heidenhain-Susa solution.

Histopathology: The endolymphatic sac and adjacent part of its duct were replaced with vascular connective tissue and new bone. The vascular connective tissue extended into the vestibular aqueduct to a point 1.04 mm. from its vestibular end. The study of consecutive sections in that area failed to demonstrate any direct communication between the endolymphatic duct and the fluid and vascular spaces of the new connective tissue and bone. There was total degeneration of the membranous vestibular and cochlear labyrinth which was presumably the result of section of the cochlear artery. The cochlear and vestibular nerves and their ganglia were completely degenerated also. There was partial resorption of the osseous spiral lamina. The fluid spaces of the inner ear were partially filled with a myxofibrous connective tissue. Rasmussen's bundle was missing.

Cat 4. Auditory conditioning followed a right destructive labyrinthotomy. An operation was then performed to remove the left endolymphatic sac and part of its duct. The auditory thresholds during the subsequent two months remained within 10 db of the preoperative level (see Fig. 3). A partial section of the cochlear nerve was then performed. After three weeks the animal was sacrificed by arterial perfusion.

Histopathology: The endolymphatic sac and adjacent part of the duct were replaced by a mass of connective tissue and new bone. Osteoid tissue extended into the vestibular aqueduct to block the end of the endolymphatic duct at a point .54 mm. from the vestibule. The bony canal of the common crus had been injured and was partially filled with new bone; however, the membranous labyrinth was intact in this region.

As a result of the operation for partial cochlear nerve section there was a diffuse degenerative change in the spiral ganglion, most severe in the apical half of the cochlea. The organ of Corti and its hair cells appeared normal throughout. The position of Reissner's membrane was normal. The vestibular labyrinth appeared normal. Rasmussen's (olivocochlear) bundle appeared normal. A moderate amount of blood was seen in the scala tympani and scala vestibuli in all three coils of the cochlea.

Cat 5. A right destructive labyrinthotomy was followed by auditory conditioning. The left endolymphatic sac and adjacent part of the duct were then surgically obliterated. There was no significant change in auditory acuity during the subsequent four months. An operation for partial section of the cochlear nerve was then performed. Following this operation there was a hearing loss involving the entire frequency range but most severe for low frequencies. Three and one-half weeks after the nerve section the animal was sacrificed.

Histopathology: The endolymphatic sac was replaced by connective tissue and new bone. Connective tissue and new bone had grown into the vestibular aqueduct to block the endolymphatic duct at a point .8 mm. from its vestibular end.

As a result of the operation for partial cochlear nerve section, there was a severe degenerative change in the cochlear nerve and spiral ganglion, most severe in the apical one-half of the cochlea. The membranous vestibular and cochlear labyrinth appeared normal. The fibers of Rasmussen's bundle were intact.

Cat 6. After a right destructive labyrinthotomy the animal was sound conditioned for auditory testing. An operation was then performed to destroy the left endolymphatic sac and the adjacent part of its duct. Auditory thresholds were normal during the subsequent six weeks. Next an operation was performed to obliterate the left periotic duct. Auditory thresholds remained normal during the subsequent two and one-half months (see Fig. 3). Then a partial section of the cochlear nerve was performed. Following the latter operation the animal sustained a hearing loss involving a wide range of frequencies but most severe for low tones. One month later the animal was sacrificed.

Histopathology: The entire endolymphatic sac was destroyed and replaced by connective tissue and new bone. New bone had grown into the vestibular aqueduct to block the lumen of the endolymphatic duct 1.0 mm. from the vestibule, (see details in Fig. 6A).

The lumen of the cochlear aqueduct was filled by a large mass of connective tissue and bone. A study of consecutive sections showed no communication between the scala tympani and the lymphatic and vascular channels of the new tissue. Camera lucida drawings and a photomicrograph of the blocked cochlear aqueduct appear in Fig. 7. The cochlear vein entered the scar tissue of the surgical defect through which it passed to communicate with veins in the mucosa of the auditory bulla. There was also a small vein which arose from the cochlear vein near the vestibular orifice of the periotic duct, proceeded along the wall of the scala tympani and passed through the margin of the round window membrane to communicate with veins in the mucosa of the middle ear.

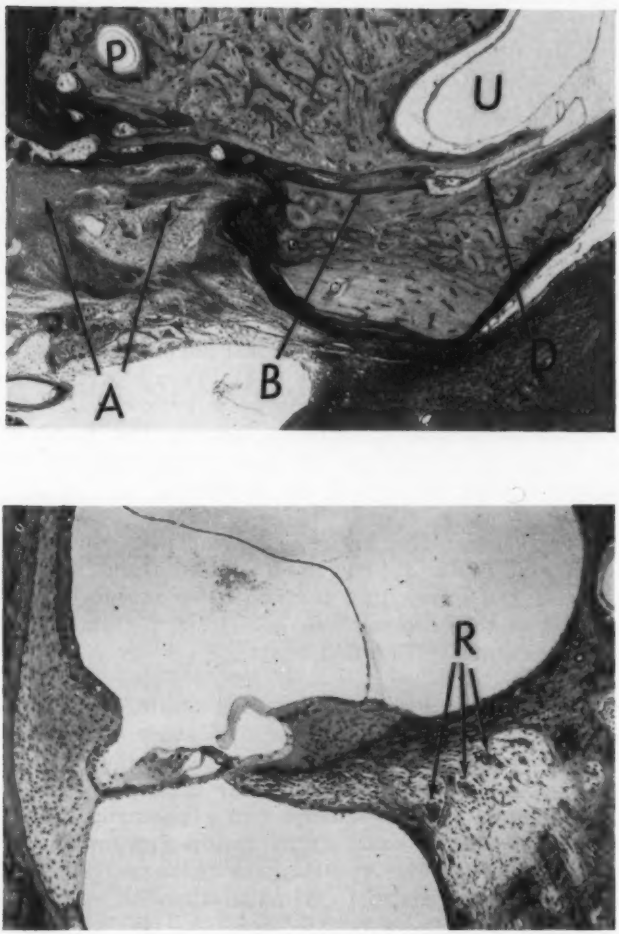

Fig. 6. Top: Photomicrograph from ear of Cat 6, showing bone and connective tissue replacing the destroyed endolymphatic sac in the area marked "A." Bone has grown into the vestibular aqueduct at "B." "D" marks the endolymphatic duct, "P" the posterior semicircular canal, and "U" the utricle. Bottom: Photomicrograph from basal coil of Cat 6. The organ of Corti is normal. Reissner's membrane bulges upward moderately. The severe loss of spiral ganglion cells and fibers is the result of the operation to section the cochlear nerve. Rasmussen's olivo-cochlear bundle (R) is intact.

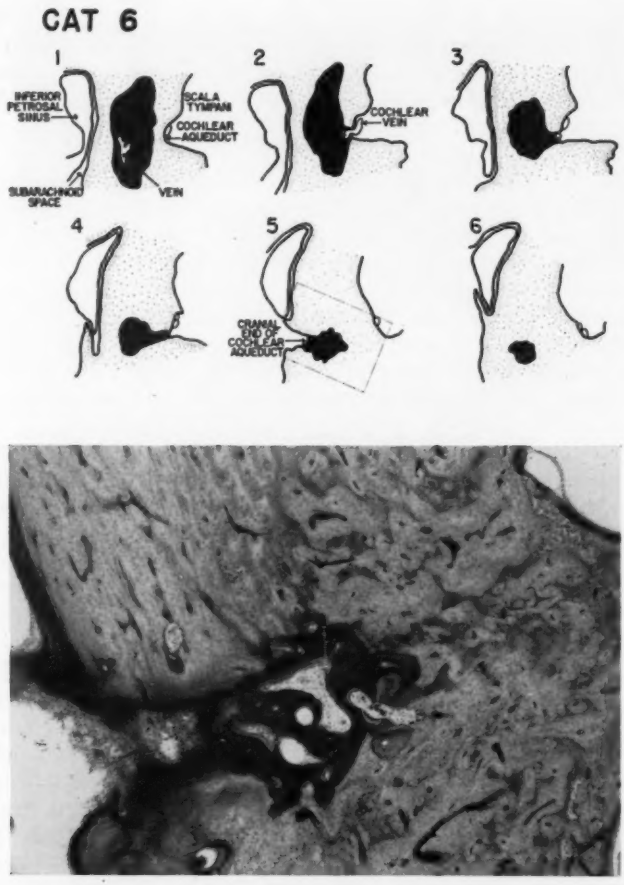

Fig. 7. Top: Camera lucida drawings from six microscopic sections in the region of the cochlear aqueduct of Cat 6. Black represents bone and connective tissue filling the surgical defect and blocking the periotic duct. The cochlear vein communicated with veins in mucosa of the auditory bulla. Bottom: Photomicrograph of No. 5 above.

Reissner's membrane bulged moderately toward the scala vestibuli. The organ of Corti appeared normal (see Fig. 6B). The vestibular membranous labyrinth appeared normal. Due to the nerve sectioning procedure there was a severe degen-

erative change in the cochlear nerve and spiral ganglion, most severe in the apical one-half of the cochlea. The facial nerves and the nerves to the superior and horizontal canal cristae were totally degenerated. Rasmussen's olivo-cochlear bundle was intact (see Fig. 6B).

Cat 7. After a right destructive labyrinthotomy the animal was conditioned for auditory testing. The left endolymphatic sac and adjacent part of the duct was then surgically destroyed. During the subsequent two months the auditory thresholds remained normal. The left periotic duct was then surgically obliterated. Following the latter procedure there was an elevation of auditory thresholds of 20 to 60 db throughout the frequency range (see Fig. 3). This hearing loss persisted until the animal was sacrificed three months later.

Histopathology: The endolymphatic sac was replaced by scar consisting of connective tissue and bone. Bone extended into the vestibular aqueduct to block the endolymphatic duct at a point 1.5 mm. from its vestibular end (see Fig. 8).

The lumen of the cochlear aqueduct was filled with connective tissue and new bone containing large vascular (venous) channels. No communication could be found between the scala tympani and the fluid and vascular channels in the scar tissue. The cochlear vein communicated with the veins in the scar of the surgical defect, which in turn communicated with veins in the mucosa of the auditory bulla and middle ear. The bony canal for the cochlear vein (which passes from the cochlear aqueduct to the inferior petrosal sinus) was narrowed by new bone formation; however, the enclosed venous channel appeared to remain as a narrowed communication between the veins in the scar and the inferior petrosal sinus (see the camera lucida drawings and photomicrograph in Fig. 9).

Reissner's membrane bulged moderately toward the scala vestibuli, particularly in the apical coil (see Fig. 8A). The organ of Corti and spiral ganglion were normal throughout. The vestibular labyrinth appeared normal.

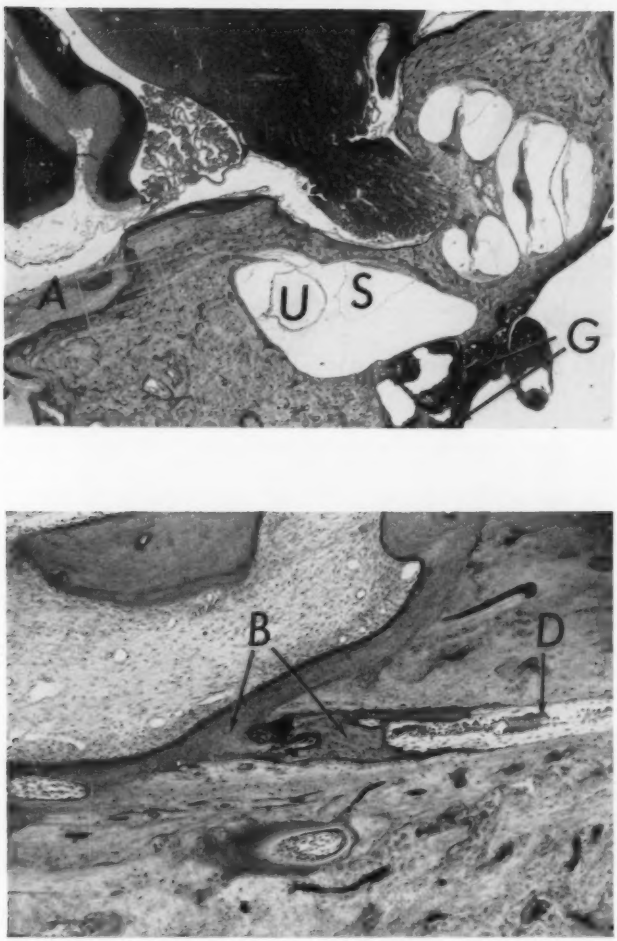

Fig. 8. Top: Photomicrograph from ear of Cat 7, showing bone and connective tissue replacing the destroyed endolymphatic sac in the area marked "A." "S" is the saccule and "U" the utricle. "G" is granulation tissue surrounding the stapes in the oval window niche. Reissner's membrane bulges upward moderately. Bottom: High power view of area marked by a rectangle in picture above. The vestibular aqueduct and its contained endolymphatic duct "D" are blocked by an ingrowth of dense new bone "B."

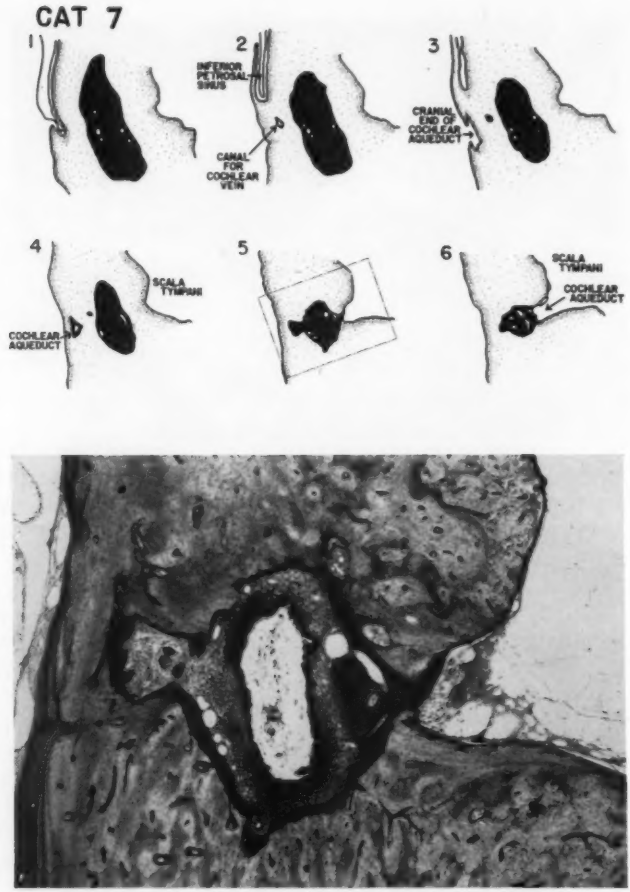

Fig. 9. Top: Camera lucida drawings to show blocked cochlear aqueduct in Cat 7. The cochlear vein communicated with the inferior petrosal sinus and with veins in the mucosa of the auditory bulla. Bottom: Photomicrograph of Drawing 5 above, showing connective tissue and bone blocking the cochlear aqueduct and its contained periotic duct.

There was a small amount of granulation tissue in the oval window niche surrounding the crura of the stapes. This tissue consisted mainly of lymphocytes and plasma cells

TABLE 'I

Cat No.	Obliteration Operations	Vestibular Upset	Hearing	Section Cochlear Nerve	Injury to Cochlear Artery	Conduction Lesion	Membranous Labyrinth
1.	Periotic duct	+	Slight loss				Serous labyrinthitis
2.	Periotic duct		Normal	+	+		Severe degeneration
3.	Endolymphatic sac		Normal	+	+		Severe degeneration
4.	Endolymphatic sac		Normal	+			Normal
5.	Endolymphatic sac		Normal	+			Normal
6.	a. Endolymphatic sac		Normal	+			
	b. Periotic duct		Normal	+			Bulge of Reissner's membrane
7.	a. Endolymphatic sac		Normal				
	b. Periotic duct		Moderate loss			+	Bulge of Reissner's membrane

densely packed into a loose connective tissue stroma; however, in one area there were also many polymorphonuclear leucocytes and several giant cells. It is impossible to know what magnitude of hearing loss, if any, could be created by this pathology. Rasmussen's bundle was normal.

A summary of the protocol as well as the functional and histological findings in these animals appears in Table I.

DISCUSSION.

Auditory Thresholds: Seven months following obliteration of the periotic duct the auditory thresholds of Cat 1 were within 10 db of normal for all frequencies except 250 c.p.s., for which there was a 15 db elevation of threshold. This cat was the only animal in the group which showed signs of an acute vestibular upset (nystagmus, ataxia) following surgery, the duration of which was five days. Histological examination of this ear revealed pathological changes consistent with an arrested acute serous labyrinthitis (see Fig. 5). The hearing of Cat 2 remained normal for a period of seven months following obliteration of the periotic duct.

The auditory thresholds for the experimental ears of Cats 3, 4, 5, 6 and 7 were normal for periods of time varying from one and one-half to four months following obliteration of their endolymphatic sacs. Histological studies showed that in each ear the endolymphatic sac was destroyed and the endolymphatic duct was blocked by connective tissue and bone.

In the experimental ears of Cats 6 and 7 the periotic ducts were obliterated at a second operation, one and one-half and two months respectively, after the operations to destroy their endolymphatic sacs. The auditory thresholds of Cat 6 were normal two and one-half months later. Cat 7, however, had a hearing loss ranging from 20 to 60 db throughout the frequency range. It is not known whether the hearing loss in this animal was due to a small inflammatory granuloma in the oval window niche or to the moderate endolymphatic hydrops existing in the cochlea. This was the only ear in the group with demonstrable lesion of the sound conducting mechanism.

Endolymphatic Sac: The endolymphatic sacs only were destroyed in the ears of Cats 3, 4 and 5. The membranous labyrinth of Cat 3 was found to be severely degenerated because of injury to the cochlear artery occurring as a result of the operation for partial cochlear nerve section. The membranous labyrinths of Cats 4 and 5, including Reissner's membranes, appeared normal after time lapses of three and five months respectively.

Bulging of Reissner's membrane appears to be an uncommon histological finding following surgical destruction of only the endolymphatic sac, occurring in only two of thirteen animals operated by Lindsay[1,4] and in neither of the two animals operated by us.

Periotic Duct: Operations to obliterate the periotic duct were performed in four ears (Cats 1, 2, 6 and 7). In the ears of Cats 1 and 2 the membranous labyrinths were damaged for reasons not directly related to operations on the periotic duct; Cat 1, because of a postoperative labyrinthitis, and Cat 2 because of section of the cochlear artery. The ears of Cats 6 and 7 had well preserved membranous labyrinths, and in both there was bulging of Reissner's membrane into the scala vestibuli. The endolymphatic sac had been obliterated previously in both of these ears. Because obliteration of the endolymphatic sac failed to produce significant histological changes in the ears of Cats 4 and 5, we tend to discount it as a cause for bulging of Reissner's membrane in Cats 6 and 7.

The significance of bulging of Reissner's membrane is difficult to evaluate. Slight displacement of the membrane in either direction has been seen in normal ears, and is thought to be an artefact of perfusion and histological preparation. Displacement of the magnitude seen in the ears of Cats 6 and 7, however, has not occurred in normal cat ears prepared by the same techniques in our laboratory.

We are aware of only one previous study concerning histological changes in the inner ear following experimental blockage of the periotic duct. This was a beautiful experiment by Uyama[5], described in the Japanese language in the Okayama Igakkai Zasshi in 1933. He used two methods for

obstructing the cochlear aqueduct in rabbits. In one group of animals sarcoma tissue (Kato lineage) was injected into the subarachnoid space of the posterior cranial fossa near the opening of the left cochlear aqueduct. Animals which lived for 15 days were sacrificed after varying time lapses. In another group of rabbits operations were performed to block the opening of the aqueduct by introducing into its lumen a small piece of dry catgut sharpened at one end. There was a striking correlation between obstruction of the cochlear aqueduct and the occurrence by bulging of Reissner's membrane. We have recorded the results of Uyama's experiment in Table II. He noted a glass-like appearance of the perilym-

TABLE II—(Uyama)

OBSTRUCTION OF COCHLEAR AQUEDUCT**

REISSNER'S MEMBRANE	None	One-Half	Greater Portion	Complete
Normal Position	2, 7, 9, 16*, 19*, 21* All controls except No. 2			
Slight Rise		1, 5, 8	3, 4,	17*, 18*
Moderate Rise		6	10, 12 Control ear No. 2	
Great Rise				11, 13, 14 15, 20*

*Catgut used for blockage. Sarcoma cells used in all others.
**Left ear of each animal was operated. Right ear used as control.

phatic fluid spaces in several ears with blocked cochlear aqueducts. Uyama failed to find evidence of labyrinthitis to explain the rise in Reissner's membrane in these ears. He made the interesting observation that the rise in Reissner's membrane was often greatest in the apical region of the cochleas. This was especially noted when the aqueduct was blocked with catgut. He theorized that perilymphatic fluid is derived from cerebrospinal fluid by way of the cochlear aqueduct and that blocking the aqueduct prevents replenish-

ment of the perilymphatic fluid which normally is slowly absorbed. Thus he believed that the endolymphatic spaces expanded to fill up the space left by the diminishing perilymphatic fluid.

Cochlear Vein: Normally most of the venous return from the cochlea passes through the cochlear vein. This vein courses along the wall of the scala tympani and enters the cochlear aqueduct. Before doing so it often is joined by a small vein which originates in the mucosa of the auditory bulla and reaches the scala tympani by passing through the margin of the round window membrane. The cochlear vein lies within the cochlear aqueduct for a distance of about 1.2 mm. At this point it enters a separate bony channel and continues within it to the inferior petrosal sinus. In four ears (Cats 1, 2, 6 and 7) the cochlear veins were interrupted and mechanically blocked with bone dust and soft tissue coincident to obliteration of the periotic ducts.

Histological examination of these ears showed that in each case drainage through the cochlear vein had been reestablished; in Cat 1 with the inferior petrosal sinus and in Cats 2, 6, and 7 with veins in the middle ear mucosa.

The absence of acute vestibular and cochlear dysfunction following interruption of the cochlear vein must be interpreted to mean that venous drainage is adequately maintained through other vessels, possibly through the communicating vein of the round window or through veins accompanying the endolymphatic sac. These findings are consistent with those of Perlman[6] who was unable to demonstrate significant functional or histological changes following interruption of the cochlear vein in guinea pigs.

SUMMARY.

Destruction of the endolymphatic sac failed to create cochlear dysfunction or histological changes in the membranous labyrinth.

Surgical blockage of the periotic duct was associated with a moderate rise in Reissner's membrane in two ears. The relationship between the interrupted periotic duct and the rise in Reissner's membrane is not distinct, because in both ears the endolymphatic sacs had been previously destroyed and also the cochlear veins were interrupted. Furthermore, it is possible that the operative procedures caused a very mild inflammatory reaction in the labyrinth of sufficient intensity to cause this rise in Reissner's membrane; also we cannot definitely rule out artefacts of histological technique as a cause for this finding. At the present time we do not believe that the evidence from our work and that of Uyamo prove that blocking the periotic duct causes dilatation of the endolymphatic system.

We do admit having a rather high index of suspicion concerning the relationship of the periotic duct to endolymphatic hydrops. To provide further information on this subject we have surgically obliterated the periotic ducts in another series of cats, leaving the cochlear vein intact in some. The opposite ear of each cat is to serve as a functional and histological control. Tests of cochlear function will be done by the method of recording evoked strychnine spikes from the auditory cortex in response to the onset of pure tones.

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A PRELIMINARY REPORT OF A NEW METHOD FOR THE EXAMINATION OF NYSTAGMUS THROUGH TELEVISION.*

JOSEPH A. SULLIVAN, M.B.,
WALTER H. JOHNSON, Ph.D.,
and
W. E. HODGES, M.A.Sc.
Toronto, Canada.

INTRODUCTION.

In order to evaluate the pathological significance of nystagmus, whether spontaneous in origin or artificially induced by stimulation in the clinic, various methods have been described. These often involve the use of mechanical devices which not only distract the patient but are also difficult to duplicate. One such earlier method utilized levers attached to the cornea or to the closed eyelid; others use optical systems pertaining to the movements of light beams reflected from mirrors attached to the eye, while recent methods record nystagmus through the electrical potentials set up by ocular movements.

Since all of these methods have certain inherent recognized deficiences, widespread attempts are being made to correct them. In England, Hood and Hallpike[1] (1953), and likewise, in Holland, Groen and Ruding[2] (1953), have made advances in the electrical potential methods by the elimination of extraneous impulses involved in the electronic apparatus. Torok[3] (1953), in U.S.A., has described a method of utilizing infrared light reflected from the patient's cornea while Pirodda[4] (1953) in Italy, has concentrated on electro-encephalographic findings for diagnosing Meniere's disease.

The entirely new method described below has certain definite clinical advantages both for the examiner and the patient. They are:

1. No equipment is attached to the patient's head.

*From the Hard of Hearing and Vestibular Research Clinics of The Department of Otolaryngology, St. Michael's Hospital, Toronto, Canada.

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2. There are no distracting influences (such as the examiner standing in line of vision, nor equipment immediately facing the patient).

3. No special precautions are required such as special illumination or electrical shielding of the patient.

APPARATUS AND PROCEDURE.

The entire recording apparatus consists of a small television camera together with a portable receiving unit (see Fig. 1). An enlarged and clear image of the patient's eye is thus recorded on the television receiver screen (see Fig. 2). The apparatus used in St. Michael's Hospital was provided through the Radio Corporation of America. The eye image as recorded is on a screen, ten inches in diameter. The camera, which can be mounted on a support attached either to the floor or suspended from the ceiling, is connected to the receiving unit by a suitable length of cable. The image of the patient's eye may be examined in another room, if desired. This method when utilized for demonstration purposes in a neighboring class room has proven to be of great merit.

The patient's eye is illuminated either by daylight or by a 60-watt lamp. The camera is placed obliquely either to one side or above the head so as not to interfere with the patient's field of vision (we find it desirable to have the patient look at a small mark on the opposite wall at a distance of at least eight feet).

By means of appropriate camera lenses, the eye can be magnified over 25 times, by filling the entire viewing area of a large screen television receiver, and still maintain a sharp image. The intensity of nystagmus may be measured by means of a suitable scale placed on the viewing screen, or, if desired, recorded photographically for future reference.

Another feature of this equipment is the fact that the examiners may be so isolated from the patient as not to interfere with the carrying out of the vestibular tests, thus assuring greater efficiency.

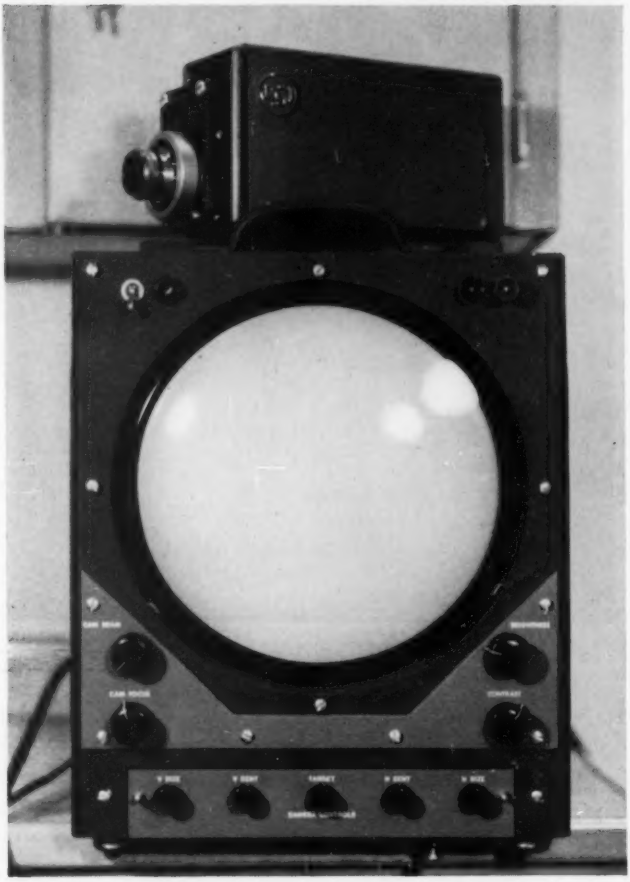

Fig. 1. Television equipment used for the clinical examination of nystagmus. The camera, a portable unit, is seen on top of the receiving unit.

SUMMARY AND CONCLUSIONS.

A new method for the clinical examination of nystagmus is described.

Fig. 2. Photograph of screen showing the image of a patient's eye. Relative magnification—approximately four times.

Its simplicity is desirable both for the examiner and for the patient; the former is freed from unstable devices, while the latter is unencumbered and at ease.

The widespread availability of the equipment and the ease and rapidity with which it can be assembled and operated are advantages worth considering.

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BILATERAL ABDUCTOR PARALYSIS OF VOCAL CORDS DURING LOCAL TONSILLECTOMY. REPORT OF A CASE AND ITS MANAGEMENT.

GEORGE S. RICHARDSON, M.D.,
Albuquerque, New Mexico.

Although there are congenital, central, peripheral, myopathic and psychogenic causes for abductor paralysis of the vocal cords, I feel that the case reported belongs in none of these. Notably it does not belong in the last, for the patient is a strong character without apparent psychodynamic influence.

Review of the literature of the past five years did not reveal a similar case.

A case was reported in the *British Medical Journal* of December 6, 1952, where a man was thought to have developed paralysis following local anesthesia but was found to have a spinal cord tumor. Another case,[1] of hemiplegia following tonsillectomy, similar to this, was reported and a case of facial paralysis during local regional (procaine) anesthesia for total tonsillectomy,[2] was also reported.

Our anesthetic in this case was NOVOL (Monocaine Hcl 1½% with Epinephrine 1:100,000—Novocal Manufacturing Company) which has been quite satisfactory in every instance before and since without evidence of toxicity. There were no systemic manifestations of toxicity such as cyanosis, bradycardia, hypotension, etc. Only sufficient 2 per cent pontocaine was used to prevent gagging. None was directed toward the larynx.

Case Report: No. 4507. Mrs. H. L. R., a 44-year-old housewife, was referred to this office because of an episcleritis and keratitis in the left eye. A possible focus was sought as her teeth had been removed previously.

Following a complete examination with X-rays of the sinuses included, the possible foci of infection lay in the ethmoids or large tonsil remnants which expressed pus.

As displacement therapy yielded no pus, the tonsil remnants were believed incriminated. A tertiary tonsillectomy was suggested.

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Her preliminary blood work and urinalysis revealed nothing startling and values were normal.

On Sept. 8, 1952, the patient entered the office for a local tonsillectomy. A preliminary single spray of pontocaine 2 per cent with application to fauces was done to lessen the gagging reflex. This was started at 7:50 a.m. with injection of Novocol from anestubes (total 10 cc.) in the usual and customary sites for production of anesthesia. Preliminary application of 2 per cent tincture of iodine was also made to the same areas two minutes before injection of anesthetic—a customary procedure in all cases. There was no outward evidence of patient excitement.

There was an immediate inspiratory stridor with coughing sufficiently violent to prevent any operative work. At 8:15 a.m. the tonsil remnants were completely anesthetized and patient complained of difficult breathing. The larynx was inspected by indirect means.

The patient volunteered that "she hadn't been able to sing for a long time." The left vocal cord was discovered lying in the midline and immovable, with some movement of the right, although this cord was traveling only from mid-abduction to the midline. A Mosher lifesaver tube was at hand. Blood pressure was 140/90. The patient was unexcited, and my excitement was not communicated to her. Intravenous Benadryl was secured.

The patient was inspected at 8:20 a.m. still in the erect position. There was no movement of either cord from midline save for movement by air. Ten mgm. of Benadryl was given intravenously. Mosher was not called upon.

There was almost immediate improvement in the respiratory difficulty. Cords were inspected at 8:30 and 8:40 a.m. At the latter time both cords were abducting with respiration and adducting with phonation. By 9:00 a.m. both tonsil remnants were removed.

The patient was placed on Chlor-Trimeton post-operatively, 4 mgm. three times daily for two days. The eye was neither sore nor red at 6:00 p.m. when seen at home. She was seen on Sept. 10, 1952, and both vocal cords were functioning normally.

The patient was last seen on June 24, 1953, neither eye nor larynx bothering her and throat without tonsil tissue. Cortone by instillation had been used intermittently following operation for month of September in affected eye.

SUMMARY.

A case is presented of apparent bilateral abductor paralysis following the use of a pontocaine spray combined with Monocaine Hydrochloride anestubes for local tonsil anesthesia. Ten milligrams of Benadryl (P.D.) injected intravenously apparently terminated the reaction and restored motility to the vocal cords.

201 Arno Street, S.E.

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PULMONARY EMBOLUS FOLLOWING TONSILLECTOMY (REPORT OF A CASE).

W. P. PIRKEY, M. D.,
Pittsburgh, Pa.

Tonsillectomy being the most frequent of operations, has had as its sequel many and varied complications. An excellent tabulation of the complications was collected by Dr. H. D. Harlowe.[1] In his paper two patients were diagnosed as having had pulmonary emboli following tonsillectomy.[2] They both were published in foreign journals and a complete description of their cases was not available at the time of this paper. This is a report of a case having a pulmonary embolus 24 hours after a local tonsillectomy treated by heparinization with resulting increased clotting time and its effect on the immediate postoperative tonsil fossa.

D.K.K., a 24-year-old white female was admitted to a local hospital on February 13, 1952, with a diagnosis of herniated intervertebral disc. On admission she was found to have chronically infected tonsils; a herniated intervertebral disc between L 2 and 3, otherwise entirely negative. No history or physical finding of phlebitis was elicited at this time.

While in the hospital undergoing conservative treatment for her intervertebral disc she developed cystitis and pyelitis. The urine culture showed hemolytic staphylococcus aureus and hemolytic streptococcus. This was controlled by neomycin. The tonsils were infected by the same organisms as those found in the urine, so it was decided to do a tonsillectomy.

On March 1, 1952, a local tonsillectomy was done without complications. There was no postoperative bleeding, no ties or sutures were placed in either fossa. The immediate postoperative results were excellent. Approximately 24 hours following the operation, while the patient was standing beside her bed, she had a sudden onset of severe pain over the right chest with associated shortness of breath and profuse sweating. Complete bedrest, oxygen, and morphine were begun immediately. Examination showed a blood pressure of 80/60, some dullness and rales over the right pulmonary base. There was no tonsil bleeding at that time. An internist was called, an ECG taken, and a diagnosis of pulmonary embolism, right lower lobe made. The patient was then placed on Depoheparin, 400 mg intramuscularly stat. A more detailed past history was obtained, which together with questionable tenderness in the left calf, made us feel that the embolism was caused by phlebitis of the left femoral vein and not by the tonsil operation. It was decided to do a femoral vein ligation as soon as the patient was able. Approximately one hour after beginning the Heparin, both tonsillar fossae began to bleed

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steadily. It was then decided to combine the femoral vein ligation with a tying of all tonsil bleeders. This was done under general anesthesia, 13 hours after the onset of the embolus. At operation, the tonsillar fossae were found to be bleeding not profusely but steadily from the entire postoperative site, both right and left. All bleeding areas were tied with No. 1 plain catgut ties. The femoral vein had been located, evidence of thrombophlebitis was found, and the vein was ligated.

Following the second procedure, the patient's recovery was uneventful. She was kept on Heparin and Dicumeral until March 16, 1952. She has had no further illness.

CONCLUSION.

A case report of a white female who had a pulmonary embolus following tonsillectomy treated by heparinization with its resulting complications has been given. Follow up on the patient has been entirely satisfactory.

1. HARLOWE, H. D. "Complication Following Tonsillectomy". THE LARYNGOSCOPE, 58:861-873, 1948.

2. a) RUEDI, L. "Unusual Complications of Tonsillectomy." *Schweiz. med. Wchnschr.*, 69:595, July 1, 1939.

b) STAMBERGER, E. "Komplikation nach Tonsillektomie auf Grund von Zirkulationsstorung", (Complications of tonsillectomy on the basis of Circulatory Lesion). *Ztschr. f. Hals., Nasen. u. Ohrenh.*, 26:442-443, 1930.

Mercy Hospital.

INFECTION FOLLOWING THE USE OF A MAGNET AS A HOLDER FOR SURGICAL INSTRUMENTS DURING OPERATION.*

ALBERT P. SELTZER, M. D.,
Philadelphia, Pa.

From personal experience, it has been found that using a magnet as a holder for small surgical instruments, in the course of a rhinoplasty, may be a source of focal skin reaction.

The magnet used (Magnagrip) is constructed of two metal trays, rectangular in shape, of sufficient difference in size, so that one fits inside the other with a narrow space between their sides.

The length of the outer tray is nine inches (23 cm.). The width is two and one-sixth inches (5 cm.). The inner tray is eight and five-eighths inches in length (20.5 cm.) and one and five-eighths inches wide (4.3 cm.). The outside depth is three-fourths of an inch (2 cm.).

Before use, the magnet is sterilized by being allowed to stand in 90 per cent alcohol, or a solution of Zephrine, for twenty minutes. There is a round opening in the floor at each end, by means of which the magnet can be secured over the chest of the patient. The magnet in this position will hold firmly all the finer instruments used, especially those used in operating on the lower portion of the nose, as in reducing the lower lateral cartilages and also on the nasal tip. It is a convenience and expedites surgery by maintaining very near to the operative field, all of the instruments used for fine detail.

The reason for the presentation of this note is to report that during a considerable time that the magnet has been

*From the Department of Plastic and Reconstructive Surgery, St. Luke's Medical Center.
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used as a matter of routine, an after effect has been observed in some cases. This effect resembled an acneiform eruption over the tip of the nose, and in one case there was a resemblance to tattooing.

When the contents of the pustules were expressed and spread on a glass slide, they were examined with a jeweler's lens. Small particles could be seen, which appeared to be minute fragments of metal, presumably steel. When tested with an ordinary magnet, they were attracted and adhered to it, suggesting that they had been derived originally from the magnet to hold the instruments.

When the magnet is not in use, it has been seen that various particles of "dust", the nature of which has not been determined, are attracted to it. This foreign material may be carried into the surgical wound on the instruments used, and so cause an acneiform infection; also, it may not be infective, but on gaining access to the skin, the particles may act as foreign bodies, causing focal increase of connective tissue, which eventually appear as minute intradermal nodules.

There is also a question, which does not appear to have had consideration. This question is, what effect, not immediate, may be the outcome of using magnetized instruments in operations on the skin?

While this idea is mentioned only as entirely theoretical, and has no necessary relation to the skin eruption described above, it might be well to consider it. There might be some relation to a similar idea, based on the study of vegetable tissue, that "the molecular movements connected with growth and the internal changes on which the activity of the protoplasm depends, whether exhibited in the formation of new cells or in motility, are probably connected with the disturbance of electrical equilibrium.[1,2]

Whether the electric properties of the magnet in question have the power of contributing to tissue changes remains to be answered.

That care should be taken to prevent the introduction of airborne particles into the surgical field by the use of the magnet as an instrument holder, is the important point of this note.

REFERENCES.

1. BARNES, A. E.: Studies on Electrophysiology, New York: Dutton & Co., 1918.

2. VAN DER WERFF, J. TH.: Biological Reactions Caused by Electrical Currents and by X-rays. New York, Elsevier Pub. Co., Inc. 1948.

2104 Spruce St.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

December 1, 1953.

Acousticon Models A-17 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.
Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Model D (Duplex); National Ultrathin Model 504; National Vanity Model 506.
Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.
Manufacturer: Johnson Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.
Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3; Otarion Model H-1; Custom "5."
Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).
Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).
Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.
Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model J-92; Silvertone Model P-15.
Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.
Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HE ARING AIDS ACCEPTED.

Acousticon Model A-300; 1 transistor, 2 tubes, and 2 batteries.
Manufacturer: Dictograph Products., Inc., 95-25 149th St., Jamaica 35, N. Y.

Audivox, Model 71; all transistor.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Maico Transist-Ear, Model O; 3 transistors and 1 battery.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis, 1.

Otarion Model C-15; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Sonotone Model 1010; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes, and 2 batteries (A & B).
Manufacturer: Telex, Inc., Telex Park, St. Paul, 1.

Zenith Model Royal-T; 3 transistors, and 1 battery.
Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

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Vice-President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Boston, Mass., May 23-24, 1954.

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Meeting: Statler Hotel, Boston, Mass., May 28-29, 1954.

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Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
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Eastern Section: Friday, Jan. 8, 1954, New York City, Waldorf Astoria Hotel.
Council Meeting: Saturday, Jan. 9, 1954, New York City, Waldorf Astoria Hotel.
Southern Section: Saturday, Jan. 16, 1954, Louisville, Ky., Brown Hotel.
Middle Section: Monday, Jan. 18, 1954, St. Louis, Mo., Park Plaza Hotel.
Western Section: Saturday, Feb. 6, 1954, Portland, Ore., University of Oregon Medical School.

(In Portland, room reservations may be made at Heathman Hotels).

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Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

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Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: Feb. 28 to Mar. 4, 1954, Mexico City.

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Secretary of Section on Otolaryngology: Max E. Pohlman, M.D.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:00 P.M., fourth Monday of each month from September to June, inclusive—Otolaryngology Section; 6:00 P.M., first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

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Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

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Meeting:

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Place:
Time:

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Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

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President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

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President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho Dr. Penido Burnier and Dr. Gabriel Porto.

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Tesorero: Dr. D. Ernesto Alonso Ferrer.

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Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

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Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

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President: Dr. Franze Conde Jahn, Caracas.
Secretary: Dr. Victorino Marquez Reveron, Caracas.
Meeting: Caracas, Venezuela, Feb. 21-25, 1954.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The following is a schedule of 1954 Meetings:

Eastern Section	Fri., Jan. 8th	New York City	The Waldorf-Astoria
Council Meeting	Sat., Jan. 9th	New York City	The Waldorf-Astoria
Southern Section	Sat., Jan. 16th	Louisville, Ky.	Brown Hotel
Middle Section	Mon., Jan. 18th	St. Louis, Mo.	Park Plaza Hotel
Western Section	Sat., Feb. 16th	Portland, Ore.	University Oregon Medical School

(In Portland, room reservations may be made at the Heathman Hotels.)

ANNUAL MEETING, Tues.-Thurs., May 25-27
Boston, Mass., Hotel Statler

May we call your attention to some of the attractions of Boston: Scene of the "Boston Tea Party" and the Battle of Bunker Hill; site of Faneuil Hall, the "Cradle of Liberty"; and the Old North Church (from the steeple of which the signal was given to Paul Revere); residences of Henry Wadsworth Longfellow, Wendell Phillips and William Lloyd Garrison; a short ride to the battle fields of Concord and Lexington, and the homes of the Alcotts, Emerson, Hawthorne and Thoreau; the birthplace of the Christian Science and Unitarian movements; and for those more interested in matters at hand, Durgin-Park for excellent food in a public market atmosphere; also the Boston Pops Orchestra for enjoyable music.

Reservations at the Boston Statler may be made by addressing Mr. Fred W. Potts, Sales Manager of the hotel.

For further information contact C. Stewart Nash, M.D., Secretary, 708 Medical Arts Bldg., Rochester (7), N. Y.

PAN-AMERICAN MEDICAL ASSOCIATION ANNOUNCES NINTH INTER-AMERICAN MEDICAL CONGRESS.

More than 2500 doctors and medical researchers from 22 nations of the Western Hemisphere will exchange information on the latest developments in medicine, surgery and related fields during a 16-day medical congress, to be held in six South American cities and aboard ship en route from New York to these ports, it was announced here today by Dr. Charles Crocker of San Francisco, Executive Secretary of the Pan American Medical Association.

This twenty-six-year-old organization of doctors, which annually brings together leaders in all branches of medicine and public health, will sail from New York January 6, with over 700 U. S. doctors and their families and associates, to meet with their colleagues at this year's conference meeting points in Caracas, San Juan, Ciudad Trujillo, St. Thomas and Havana, Dr. Crocker added.

The scientific sessions will include allergy, anesthesiology, broncho-esophagology, cardio-vascular diseases, dentistry, dermatology, diabetes, endrocrinology, forensic medicine, gastro-enterology, general surgery, geriatrics and gerontology, gynecology and obstetrics, hospital administration, industrial and traumatic surgery, industrial medicine, internal medicine, medical jurisprudence, medical education, military medicine, neoplastic diseases, neuropsychiatry, neurosurgery, ophthalmology, oral surgery, orthopedic surgery, otorhinolaryngology, pathology, pediatrics, pharmacology and new drugs, plastic surgery, public health and preventive medicine, pulmonary diseases, proctology, physical medicine and rehabilitation, roentgenology, rheumatic and arthritic disorders, therapeutics

and nutritional research, diagnostic roentgenology, thoracic surgery, tropical medicine, and urology.

Papers in these fields will be presented each morning of the Congress, on ship and ashore. The afternoons will be occupied with panel discussions, presentations of scientific exhibits and the showing of sound and color films concerning new techniques and recent results of clinical and laboratory research.

Special sessions concerning "The Problem of Nutrition in the Western Hemisphere" will be conducted in Venezuela and Cuba, led by Dr. E. V. McCollum, of Johns Hopkins University, and Dr. Arthur Lejwa, of Long Island University. Other special sessions will be held in La Guaira, San Juan, Ciudad Trujillo and Havana.

Dr. Albert H. Andrews, Jr., clinical assistant professor of bronchoesophagology in the Department of Otolaryngology, University of Illinois, has been elected president of the Otolaryngologic Alumni of the University, and Dr. Burton J. Soboroff, was elected secretary-treasurer at the meeting of the Society held in Chicago, October 12, 1953.

Dr. Francis L. Lederer, of Chicago, was named outstanding teacher of the year 1952-53 by the Otolaryngological Alumni Association of the University of Illinois. He was presented with an engraved silver cigarette box.

DR. HALLOWELL DAVIS WINS OTOLOGY PRIZE.

Dr. Hallowell Davis, director of research at Central Institute for the Deaf, St. Louis, has been awarded the George E. Shambaugh prize in otology—by the Collegium Oto-Rhino-

Laryngologicum Amicitae Sacrum. He is the first American scientist to receive the honor.

Dr. Davis was presented with $1000 award "as a recognition for outstanding work on the physiology of hearing and the development of testing procedures for hard of hearing individuals." The Collegium is an international society devoted to scientific study of the ear, nose and throat.

The Shambaugh prize, established in 1947, is awarded every three years to the scientist selected as having made an outstanding contribution to knowledge of hearing and deafness.

NINETEENTH INTERNATIONAL CONGRESS OF OTO-NEURO-OPHTHALMOLOGY.

The Nineteenth International Congress of Oto-Neuro-Ophthalmology will be held on the occasion of the commemoration of the foundation of São Paulo, from June 11 to 17, 1954, in the city of São Paulo (Brazil).

The official themes are:

1. Metabolical and avitaminotic disturbances in oto-neuro-ophthalmology.
2. The physiopathology of the facial nerve.

These themes are subdivided into various topics, and official speakers from different countries have been invited to prepare special themes.

The official languages are: Portuguese, Spanish, English and French.

All physicians, specialists in oto-rhino-laryngology, neurology and ophthalmology may apply for membership.

Prof. Cyro de Rezende is President of the Congress, and Dr. Luiz Piza Neto is general secretary.

Inquiries or suggestions about the Congress should be addressed to: Clínica Oftalmológica, Hospital das Clínicas, São Paulo, Brazil.

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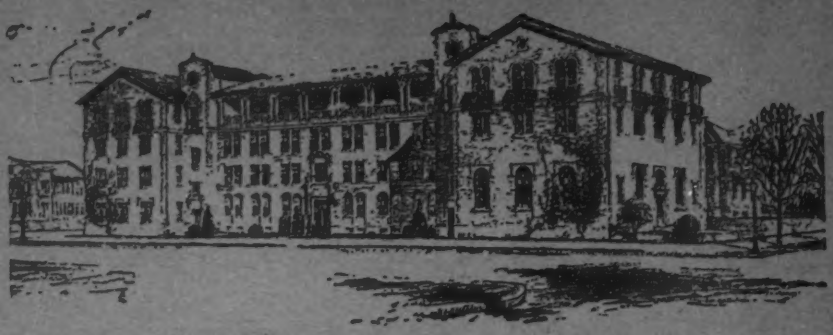
September through June

*Our subscribers are asked to send us information on other institutions giving such courses.



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